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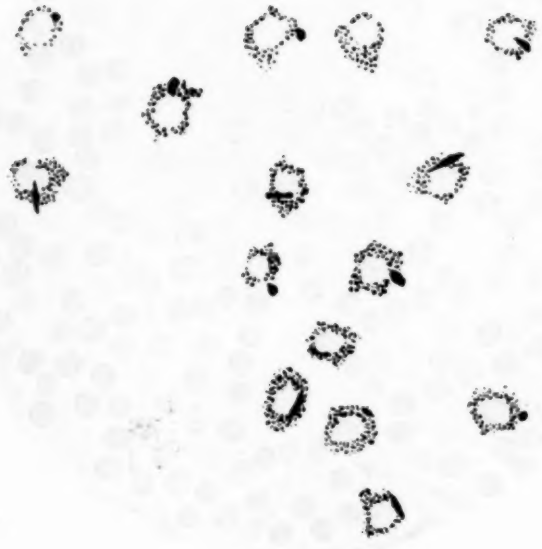

SPINDLE LIKE FORMATIONS FOUND IN UVEAL TISSUE IN SYMPATHETIC OPHTHALMITIS, SHOWN PRINCIPALLY EMBEDDED IN FUCHSINOPHILE CELLS. (ICHIKAWA.)

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SPINDLE LIKE FORMATIONS IN THE INFLAMED UVEAL TISSUE OF SYMPATHETIC OPHTHALMITIS.

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These observations, reported from the University Eye Clinic of Kioto, call attention to a class of bodies that have hitherto received little attention from histologists, but are probably crystals and of considerable importance. With one colored plate. Translated from the German by H. W. Aufmwasser, M. D.

Of late I have been able to demonstrate in bacteriologic investigations of sections of eyes enucleated for sympathetic ophthalmitis, a spindle-shaped formation in the inflammatory uveal tissues. As this remarkable occurrence has not been previously observed by any one, I wish to report about it in the following:

Siegrist,[1] Manolescu,[2] Quint,[3] Calhoun,[4] Chaillous,[5] Browning,[6] and others have noted the favorable effect of salvarsan in sympathetic ophthalmitis. This therapeutic experience has brought authors to believe that the exciting cause of this illness may be in close connection with the exciting cause of syphilis. This seemed to be more probable, since Gradle[7] could prove in three cases of perforating injuries of the eyes, the presence of a spirillum.

I have, therefore, in my investigations made it my task to prove the correctness of this conception. The eyeballs which I had for my experiments were seven in number. For the identification of the spirochetes we used the method of Levaditi. Among these seven eyeballs, six were already embedded in celloidin, so that these had to be freed from celloidin for the Levaditi treatment. They were all sympathizing eyes and have shown the typical picture of sympathetic ophthalmitis.

The result of this examination was, however, wholly negative, altho I examined the sections very carefully.

Since my first examination was without result, I tried then to find any bacteria in the sections of the sympathizing ophthalmitis.

That the histologic picture of this affection is very similar to that of tuberculosis, is sufficiently known. No one has succeeded so far in proving the presence of tubercle bacilli in the inflamed tissue of sympathetic ophthalmia. Neither have the numerous tests by implantation on animals susceptible to tuberculosis furnished a positive proof. The search for bacteria of other nature was also negative.

As matters stand, the finding of bacteria in the tissue of sympathetic ophthalmitis is therefore an exceedingly difficult problem. In spite of all this, the fact that no one used the method of Ohtori[8] of staining, for the bacteriologic investigation of sympathetic ophthalmia, caused me to stain these sections by this method.

I have stained by this method numerous sections which had been kept in the clinic for a long time. In the beginning the results were entirely negative. Occasionally, however, I have found in one of the sections, which was treated for a short time with acid, a highly remarkable fact, a fact which no one had so far observed. That is

the occurrence of peculiarly formed fuchsinophile structures in the inflamed uveal tissue. I wish, therefore, to report more minutely about these structures.

MORPHOLOGY.—It concerns a spindle-like formation whose size fluctuated. In length it measured 6.64μ to 2.82μ, in thickness 1.0μ-0.55μ. Most of these were pointed at both ends, while others ended more or less bluntly. Generally the short specimens were thicker and less pointed than the long ones. They have mostly a straight, elongated form; those which were bent were very few. Its surface is everywhere smooth; in no place angular. One sees in places, taken up in eosinophile leucocytes, rounded formations which in thickness and color resemble perfectly the formation in question, and which cannot be regarded as anything else but the spindles seen in the section transversely. The formations in question are consequently, according to their shape, to be likened to a true spindle. They appear in sections stained with fuchsin with few exceptions, spotted-like, as they are at times less stained in the center or at one or the other end, about which I will speak later. (See Plate I.)

STAINING CONDITIONS. — They are fuchsinophile, not acid-fast, but alcohol-fast; in long exposure to acid they lose their stain quickly. They are stained slightly rose with eosin, and also with Giemsa; but these two staining fluids give no contrast staining. Neither methylen blue nor Gram's give a positive result.

The best method that I have found so far is the following:

1. Staining with carbolfuchsin for three or four hours. (Better in incubator.)
2. After rinsing in distilled water for five or six seconds, stain in the following solution:

Saturated solution of picric acid	100.0
Indigocarmin	0.5

3. Wash in distilled water.
4. Differentiation in absolute alcohol.
5. Xylol balsam.

This method is in principle similar to Ohtori's method for staining tubercle bacilli. The difference consists only in the fact that by my method the treatment with hydrochloric-acid-alcohol is superfluous.

In successful staining the spindles stain with fuchsin red, collagenous tissue and nucleus with indigocarmin green, red blood cells reddish-yellow. Besides the spindles, the granules of the eosinophile leucocytes and Russell's bodies, should they be present, are also stained red. The red stain of the spindles and of the eosinophile granules is, however, not altogether the same, since the latter are distinctly darker stained than the former. (See Plate.)

The red stain of the spindles is generally not a proportionate one; they are in places stained weaker, so they appear more or less spotted. The pale stained portion, which can make its appearance anywhere, appears then as a shadow. The spindles, which on both ends have such weaker stained portions, make one think of a rod.

Heidenhain's iron-alum-hematoxylin method also gives a beautiful stain; the spindles with the eosinophile granules and nuclei are stained intense black.

BIOLOGIC CHARACTERS.—Histologically we notice above all that the spindles in the tissues are in intimate connection with eosinophile leucocytes. They are found most regularly within the bodies of the eosinophiles; few are found external to the latter. This condition facilitates exceedingly the discovering of the spindles in the tissues, especially when they are only sparingly present.

They are found exclusively in inflamed uveal tissue, and indeed appear often in the vicinity of the giant cells, where the eosinophiles tend to assemble. They always lie singly, and are therefore easily overlooked.

CHEMICAL CHARACTERS. — They are soluble in ether-alcohol, likewise in acid and water. I have placed the sections, after freeing them from celloidin, for 12 hours in an acetic solution, and in boiling water for 15 minutes, without, however, being able to notice a marked difference in the numbers as well as in the staining quality of the

spindles. In warm one per cent caustic potash solution they become unstainable after five minutes. I have furthermore treated the sections freed from celloidin with antiformin; the spindles behaved the same as with the treatment with potash solution, as not antiformin-fast.

FREQUENCY OF THE SPINDLES.—In order to know how often the spindles appear in sympathetic ophthalmitis, I have examined all the sections which had been kept in the clinic. With these I had at my disposal seven cases which all showed typically the picture of the disease. Among these seven I could prove the presence of the spindles only in five.

The number of spindles differ as to the case. Among five cases with positive findings I could in two cases find a moderate abundance, in others only a scarcity of spindles. When they are numerous their discovery is not difficult. I could, in one case which contained them most numerously, observe in one field 8-10 spindles. If they are sparingly present their discovery is a very painstaking task, so that after repeated examinations of a whole section we find only a solitary spindle. As to the presence of spindles in other inflammatory eye affections, I wish to remark here, that I have in no case observed them. As a control experiment I have examined three cases of phthisis bulbi dolorosa, three cases of nonsympathetic uveitis after perforating injury, and one case of tuberculosis of the iris and the ciliary body. In all these cases the result was negative. From my examinations up to this time, spindles appear only in sympathetic ophthalmia.

What are these spindles? Primarily one asks himself the question if they are not artificial products which through fixation and hardening have originated secondarily. To this I wish to remark at once that from the five eyeballs which have shown the positive test, four were fixed in Mueller's solution, and the other in the Orth-Mueller solution, immediately after enucleation. Of two eyeballs in which the test was negative, the one was fixed in Zenker's solution, the other in formalin. Undoubtedly the almost regular appearance of these in eosinophile leucocytes, if they can be found at all in the tissues, speaks decidedly against this conception that they are artificial.

That they are neither tubercle bacilli nor similar bacilli can hardly be doubted by their behavior towards acid and their external shape.

Are they not a crystal? According to their external appearance they resemble a crystal very much. It requires hardly further explanation that they are neither fat-crystals nor fuscineedles. The intimate relation which exists between the spindles and eosinophiles permits the conjecture that they have been formed thru melting together or crystallization of the eosinophile granules. This conception is so much more probable since, according to some authors, the so-called Charcot-Leyden crystals are believed to be crystallization products of these granules. Levy[9] could find, for instance, in 47 cases of nasal polyps 32 times the Charcot-Leyden crystals and 17 times in 29 cases in such individuals, who never had suffered from asthma. The crystals thereby have shown in their staining qualities a pronounced acidophile condition. They have also been in the tissues with the eosinophile leucocytes, with which they were numerously associated and intimately connected. Levy also remarks that the crystals are found to be more numerous when the tissue which is under investigation has been exposed primarily to spontaneous putrefaction.

My spindles and the Charcot-Leyden crystals are very similar in their staining conditions, as both are decidedly acidophile. Whether they act chemically alike is for the present not ascertainable, as the behavior of Charcot-Leyden crystals in hardened preparations had not heretofore been mentioned in the literature, as far as my studies are concerned. The Charcot-Leyden crystals are in fresh condition very easily soluble in acid, alkali and warm water.

Lubarsch[10] was also able to prove in the cells of the human testicle, small crystalline bodies, which were in hardened preparations soluble in boiling water and 50 per cent acetic acid, but insoluble in alkali. My spindles differentiate, therefore, from these small bodies thru their chemical behavior.

Are they not a kind of microorganism? The pathogenic bacilli, named *bacillus fusiformis,* have a spindle shaped form, and have been found heretofore in different diseases both local and general. The most important form of these spindle-like bacilli is the bacillus Vincenti and the one found by Babes[11] in scurvy. Similar bacilli are encountered also occasionally in the normal buccal cavity. All these bacilli distinguish themselves[12] thru their peculiar form, their spotted appearance in stained condition, and finally thru their inconstant size. They have, therefore, no similarity at all with my spindles, at least not in their external form.

That the spindles are not microorganisms is undoubtedly true. In the same manner there is no certain proof for their nature of a microorganism. To decide this question we have to wait till the time comes when their multiplication can be surely proven in some nutrient medium. The differentiation of the spindles from Charcot-Leyden crystals must also be left for future investigations.

The fact which, however, deserves special consideration is that the spindles are found very frequently only in sympathetic ophthalmitis. If these spindles were a kind of spindle-formed bacilli, and would appear sometimes as a saprophytic inhabitant of the normal buccal cavity, it would be not at all unnatural, that they would occasionally, in a perforating injury of the eye, infect the wound and produce sympathetic ophthalmitis.

While the true nature of my spindles is not at all clear at this time, and I certainly do not intend to assert that they are of any importance for the etiology of sympathetic ophthalmitis, I claim the privilege of now bringing to the knoweldge of the profession the result of my investigations, which at any rate are remarkable in their findings.

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CONGENITAL TUMOR OF THE CORNEA OF DOUBTFUL CLASSIFICATION.

H. H. Stark, M. D.

EL PASO, TEXAS.

The case here reported seems unique in that the probable dermoid tumor was nowhere connected with the limbus. The reports on its pathologic anatomy and review of the literature bearing most directly upon it complete the paper. With four illustrations.

Uncomplicated tumors confined to the cornea, either congenital or acquired, are of so rare an occurrence that I feel the case I have recently seen may be worthy of reporting.

CASE.

A. A., Mexican, female, age 15; well nourished but of nervous temperament, reported to me on November 6, 1917, having been sent home from school by her teacher with instructions to see a doctor regarding a growth she had on her right eye, which she claimed had been there since birth. No family history could be obtained, both parents being dead.

On examination the following conditions were found:

Both lids were normal in shape and movement. The right eye was slightly smaller and deeper set in the orbit than the left, having a convergent strabismus of about twenty degrees, with no movement to the temporal side past the median line. The conjunctiva and sclera were apparently normal.

On the cornea was a growth protruding between the lids, with a perfectly round base of about eight millimeters in diameter, and about five millimeters high. It was bluntly conical in shape, and slightly flattened, horizontally, from lid pressure. The body of the tumor was light pink, the tip darker in color with faint stippling due possibly to exposure and inflammation. On examination with a loupe the stippling became faintly papillary in character, but no hairs could be seen.

Surrounding the tumor was a distinct area of clear cornea about three

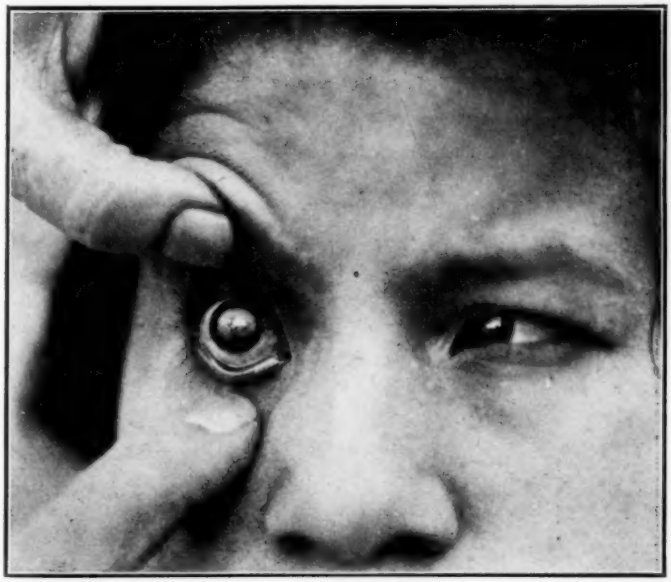

Fig. 1. Congenital tumor of cornea, front view showing separation of tumor from the limbus.

millimeters wide on the temporal side, shading down to two millimeters on the nasal side, having what appeared to be a normal curvature. On the nasal side with the aid of a loupe could be seen one large, one medium and several finer vessels passing thru the cornea from the limbus, terminating in the tumor, but no clouding of the corneal tisue. Thru the clear cornea could be seen the iris, normal in color. The pupil was dilated evenly with atropin, and with an oblique view showed normal reflex of the fundus.

An interview with a relative with whom the patient was living confirmed the history that the tumor was congenital. She also said that it had increased in size during the last few years.

Photographs of the eye were taken, and a few days later, under a general anesthetic, the growth was removed. On picking up the tumor with forceps it was found to be very hard, requiring a firmer grip than ordinary tissue. After fixing, a cataract knife was passed along the base of the tumor at its juncture with the cornea, carefully working and pulling on the tumor, thinking there might possibly be a connection some place with the anterior chamber.

After loosening part of the base and pulling with considerable force, a distinct line of demarcation could be seen between the tumor and cornea, along which was passed the knife, easily peeling the growth from its attachment. About the center of the base of the tumor was a marked bleeding, coming from a point in the cornea. The bleeding continued for several minutes after the removal of the growth.

Examination of the cornea after the operation showed it to have a normal curvature and apparently a normal thickness and transparency. There was no sign of scar tissue or injury in any place. The healing was rather slow on account of the large area to be covered with epithelium. The spot in the center corresponding to the bleeding point remained red for at least two weeks, as though a blood clot had been formed, finally disappearing. At the time of the operation, blood was taken for a Wassermann test, which proved negative.

After healing, the pupil was dilated with atropin and an indistinct view of the fundus was obtained, but no abnor-

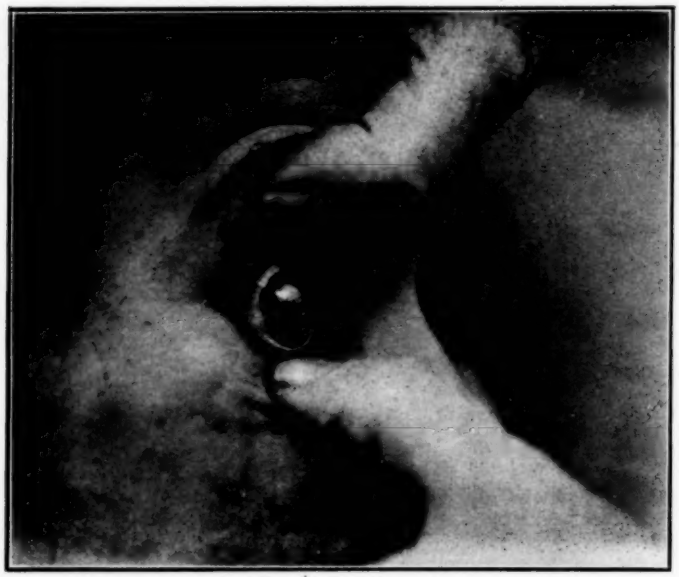

Fig. 2. Tumor of cornea from the side showing protrusion of tumor.

malities were found. The cornea at the present time shows a thick mottling of scar tissue rather than a dense scar, many places being so clear that a distinct view of the iris may be had. The eye was practically blind, no doubt from the fact that vision had never been developed. Now, seven months after the operation, there is no recurrence. The left eye was normal in appearance and vision.

EXAMINATION OF THE TUMOR.

Much to my regret, I allowed eagerness on my part to determine the nature of the growth to prompt me to turn the tumor over to our local pathologist, with instructions to make frozen sections rather than to have serial sections made. The frozen sections were made from the center of the tumor and stained with hematoxylin eosin. Examination showed the tumor not to be of ordinary character and some of the millimeters in diameter and 2 or 3 millimeters thick, covered on one side with epithelium, the other side being the raw surface where it was peeled off the eye. The epithelium was roughened in places, containing elevations that might be classed as small papillae, but for the most part it looked as if it had been subject to considerable ulceration, with an attempt at healing. The whole specimen was hardened in formaldehyde. A small section was taken out through the center for freezing to section.

"On section the tissue was very firm, and the frozen block cut with difficulty, like cutting a piece of tough leather. Sections stained in hematoxylin and eosin, on microscopic examination showed a layer of fibrous tissue, covered by a fairly thick layer of stratified epithelium. The epithelium was somewhat irregular on the surface, and in the space beneath it there was considerable infiltration with small round cells. In none

Fig. 3. Eye after removal of tumor. Front view.

sections were sent to different pathologists for their opinions. Later, a part of the tumor was embedded in paraffin, sectioned and stained with van Gieson's stain and sent to the same men, these procedures using about two-thirds of the tumor. The remaining one-third was embedded in paraffin and sectioned serially.

Report of Dr. W. W. Waite, El Paso, Texas.—"I wish to report on the pathologic specimen removed from the eye of a Mexican girl. This specimen was a small button or disc-shaped tumor, 8

of the sections was there any suggestion of glands or hair. Plate II.

"The connective or fibrous tissue layer was made up of rather coarse fibres, stained deeply with eosin, in some ways suggesting smooth muscle tissue, but further examination showed there was considerable hyalin change. In the fibrous tissue layer there were small areas of fatty tissue. The fact that hyalin degeneration was present in the fibrous tissue shows that it was degenerating. To make sure that the fibrous tissue was really connective tissue, sec-

tions were stained with van Gieson's connective tissue stain, and the entire layer took the red stain. Another piece of tissue was embedded in paraffin and sectioned. The results from these sections were the same as those described above. In looking over a large number of sections, I did not see anything that resembled a gland or hair-follicle. My diagnosis of this case would be a *fibroma.*"

Report of Dr. F. H. Verhoeff, Boston, Mass.—"The two sections submitted to me show the tumor to consist chiefly of hyalin fibrous tissue. Its surface shows marked papillary outgrowths and is covered with squamous epithelium, beneath which in places there is a moderate infiltration with chronic inflammatory cells. At the base of the tumor there is an abundance of adipose tissue. Diagnosis —*dermoid tumor* of the cornea.

"The unusual feature of this case was the marked character of the surface; and the fact, apparently indicated by the photograph of the patient, that the tumor was not connected with the limbus, but was confined to the cornea. There are no sweat or sebaceous glands in the section, but these are not necessary for the diagnosis. It is possible, however, that other sections may show such glands. I have seen one similar case, in which, in addition there was an independent teratoid tumor of the bulbar conjunctiva." In a subsequent letter he states: "The large amount of fat tissue present in the other sections you sent me rules out a simple fibroma. You could call this tumor a dermo-fibro-lipoma if you wished, but it is essentially of the same nature as a dermoid."

Report of Dr. Allen J. Smith, Pathological Department, University of Pennsylvania.—Through the kindness of Dr. William Zentmayer, sections of this tumor were submitted to Dr. Allen J. Smith, Pathologic Department, University of Pennsylvania, who writes: "I must confess to an inability to recognize the tissues as corneal had I not been told of their origin. The epithelium is too thick, forms penetrating roots or pegs, and in its best places is not flat enough. I cannot distinguish a Bowman's membrane; the matrix is not lamellated, is too cellular, and in its vessels (of course, because of the fact the sections do not reach all the way through the cornea), I find no Descemet's membrane in its lining cells. True, too, in several of the sections, fat cells, as in isolated cells, occur in the matrix.

Fig. 4. Eye after removal of tumor. Lateral view.

"The cornea apparently had small nodosities or granular projections upon its surface, as small fibricular protrusions appear in the section covered by epithelium. The thickened epithelial layer, such nodosities, even the epithelium penetrations, and, of course, the fibrous tissue, especially the finer parts close to the surface, might well be explained as to the vascularization referred to, on the basis of a chronic inflammatory process, but I can scarcely think of the adipose cells in this relation. I feel this feature can only be explained by the suggestion

FIG. 5.—SECTION OF TUMOR SHOWING CONNECTIVE TISSUE MASSES AND FAT CELLS IN STARK'S CASE OF TUMOR OF THE CORNEA.

FIG. 6.—SECTION OF TUMOR OF CORNEA (STARK'S CASE) SHOWING PAPILLAE ON PART OF THE SURFACE.

you give me that the condition is a dermoid.

"With this idea that I have skin instead of, or added to corneal tissue, the thickening of the epithelium, the non-lamellated fibrous matrix, the presence of blood vessels, and particularly the presence of fat, are all in conformity, but I wish Dr. Stark could also find a hair or two or a skin gland in some of the material to complete the picture. I am inclined to agree very strongly that this is a dermoid, but I would urge further search for one of these structures as final proof, although I do not feel that failure to find one or the other bars the diagnosis, which stands more as the most probable condition, at any rate. I feel that a chronic inflammation has, however, co-existed, the swollen epithelial cells, the epithelial hyperplasia in producing the penetrating roots, the turgid capillaries and the cellular investments along these and some of the older vessels, and the dense fibrous tissue areas met with, I think, argue for this, and doubtless this has occasioned a little nodular papilla as well. I do not seriously question the *diagnosis of a dermoid,* but feel that he may be able to establish the point beyond the shadow of a doubt if he will continue the examination of the material."

Dr. Marcus Feingold, New Orleans, La. "The specimens submitted represent perpendicular sections through a meniscus-like growth of about 7 by 1.5 mm. The mass of the growth is made up of coarse bundles of connective tissue, closely packed and running in different directions, containing very few cells and blood vessels. It is covered by an epithelium similar to corneal epithelium, but containing decidedly more layers; no cornification can be seen anywhere. The epithelium runs smoothly over the growth but forms short down-growths and thickenings here and there to compensate for the slightly irregular surface of the underlying tissue. The epithelium is overlying the connective tissue directly; only in some short places is a more loosely arranged connective tissue interposed.

In a very few sections typical fat cells can be seen near the wound-surface of the growth, and extending from it into some spaces between the connective tissue. In a small area at the edge of the growth a decidedly papillary formation of connective tissue and epithelium is seen (Plate III), with arrangement of the blood vessels typical for it. Here some infiltration with cells is visible, the nature of which cannot be decided owing to the defective preparation. No satisfactory study of the connective tissue is possible.

The growth represents, therefore, a connective tissue tumor covered by epithelium; and may be considered as the result of scar formation following an intrauterine inflammation of the cornea or possibly as a product of adhesion between amnion and cornea."

After receiving these reports, serial sections of the remaining one-third of the tumor were made and carefully examined, all showing the same character of tissue as was found in the other part of the tumor. No signs of hair follicles or glands was found, nor do I believe there were any in the other sections.

CORNEAL TUMORS IN GENERAL.

Tumors of the cornea are divided as other tumors into malignant and benign, the malignant being sarcoma and epithelioma; the benign being fibromyxoma, papilloma, dermoids and teratoids.

In considering corneal tumors, Parsons[1] says:

"A review of the recorded cases of corneal tumors leaves one in great doubt as to their true status. No sharp line can be drawn between purely inflammatory conditions and many so-called growths. There is an even gradation from the simple corneal scar to the myxoma on one hand and the papilloma on the other. . . . The same applies with equal force to the new growths, both benign and malignant, for all are vascular, though in varying degrees, and derive their vessels from the limbus. Most doubtful of all are the malignant growths, especially the epitheliomata and the endothelial type of sarcomata."

He is justified in his contention that growths of any kind confined to the cornea are rare, as it is a well-known fact that the favorite seat of practically all tumors is found, on careful reading of

the literature, either astride the limbus or connected with the limbus in some way. This fact becomes more evident when one makes a search of the literature. In reviewing different articles on tumors of the eye, it was found difficult in many cases to determine from the description whether the tumor was confined to the cornea or had some connection with the limbus. It is true that only a slight connection with the limbus is necessary, because, as the growth increases in size, advancement is usually made over the corneal surface.

Many cases of reported corneal tumors are found, on careful reading of the article, to have connection with the limbus. Even many of those looking like pure corneal growths are questionable and should not be so classified. However, including doubtful cases, the number of tumors confined to the cornea is very small, in comparison with all tumors of the eye. A fairly good search of the literature was made classifying the tumors under the head of malignant growth, including sarcoma and carcinoma, papilloma, fibromyxoma, dermoids and teratomas, the result being as follows: malignant growths, 20; papilloma, 4; fibromyxoma, 1.

Of dermoids and teratomas there was none, unless we include multiple tumors of the eye, where one was situated on the cornea, the second on some other part of the eye. Even these are very rare. Just why there are so many more malignant than benign growths is a doubtful question. Two views may be taken on this point—first, that many of the malignant growths are simply degeneration of a former benign tumor; second, that the tendency is stronger with the oculist to report a malignant growth than one of a benign character. No doubt both of these views have a bearing on the question.

In considering this tumor, we may safely exclude malignancy. The history of the case and the histologic structure of the growth would exclude papilloma, leaving us only the dermoid and fibroid to be considered.

DERMOID TUMORS AND THEIR ORIGIN.

According to Parsons[2], "Dermoids are lenticular yellow or reddish congenital tumors, which occur astride the corneal margin, usually on the outer side, containing the elements of skin (stratified epithelium, hairs, sebaceous glands, etc.). They were described as early as 1742, and four cases were published by Wardrop in 1808. They were named 'dermoids' by Ryba in 1853. While commonest at the outer part, they occur rarely at any part of the limbus, even under the outer canthus. They are often associated with other congenital malformations—(according to Picqué, 27 out of 94)—colobomata of the lids, fleshy bands from the globe to the face, etc."

Various theories have been suggested for the explanation of dermoids. Ryba suggested failure of complete closure of the lids, with consequent cornification of the conjunctiva, and cited the correspondence of dermoids and lid-notch. This view is accepted by Bland-Sutton and Treacher Collins; the latter regards cryptophthalmia, in which the whole surface of the eye-ball is covered with skin, as the ultimate manifestation of dermoid development. Gallenga drew attention to the plica semilunaris, which in fetal life, covers the globe like its phylogenetic equivalent, nictitating membrane. He thought it might remain adherent to the membrane. Osborn thought the tumor might be a remnant of the epiblast which forms the lens. Van Duyse considered it was due to an adhesion of the amnion to the eye. While it is not the province of this paper to discuss the origin of these growths, it is worth considering, as it may be possible that both the theories of Ryba and van Duyse are correct; and that those showing true dermal character come from the lid, while those without the dermal characteristics come from the amnion.

CLASSIFICATION OF DERMOID TUMORS.

On the classification of these tumors many interesting and instructive papers have been written. Among the early ones is Gallenga[3], who classifies them as, first: pure dermoids; second, lipo-dermoids; third, mixed form or lipomata containing acinous or rather acino-tubular glands, or cartilage. Concerning the origin of the pure typical dermoids, Gallenga accepts Ryba's explanation, according to

which dermoids develop as the result of disturbed closure of the oblique facial fissures, and thereby also of the palpebral gap.

Saemisch[4] says that, "the dermoid of the conjunctiva is the designation usually applied to tumors which are congenital and closely related to the skin in their histologic features. They are accordingly to be interpreted as teratoid tumors. However, the histologic components of the skin may be unequally developed in individual cases, the epidermoidal structures of the skin, the hairs as well as the glands (sebaceous and sweat glands) being much more distinctly manifested in some cases than in others. The degree of development of the subcutaneous fatty cellular tissue is especially variable, being sometimes merely suggested, whereas on the other hand it may form the main bulk of the tumor. Nerves, vessels, and smooth muscle fibres are likewise seen variably developed in their growths; more rarely discs of cartilage have been noted. . . . Notwithstanding the great variability in the histologic behavior of these tumors, they share the common peculiarity of representing cutaneous structures which have been developed in the conjunctiva, although the individual tissue varieties of cutaneous character are by no means necessarily developed to the same degree. Hence it does not appear to be justifiable to select this factor as the criterion for a classification of these tumors, as suggested by Gallenga, but it would seem more appropriate to have the classification of these tumors on their topography."

Fuchs[5] says: "A dermoid tumor is a flat growth of a solid consistence, which, so to speak, straddles the margin of the cornea, being situated partly in the conjunctiva and partly in the cornea, with which latter it is immovably connected. It most frequently occurs on the external (temporal) side of the cornea. Histologic examination shows that the growth possesses the composition of the external skin. It consists of a stroma of connective tissue, covered with epidermis, and contains hair follicles and various glands. It is, so to speak, an island of skin upon the surface of the ball."

The latest and perhaps the most exhaustive paper on dermoids is that by Pignatari[6], who writes as follows:

"As shown by numerous reports in the literature concerning dermoids of the corneo-scleral junction, there is not one constant structure for these mixed teratomata. According to Gallenga's classification, which is adopted by the author, these growths are divided into several groups, as follows:

"A dermoid of the conjunctiva is a congenital tumor whose histologic structure has very close relations with that of the normal skin. Its volume is usually small, seldom more than half a nut; the color is usually whitish, often slightly pink or yellowish, a tint attributed formerly to the presence of fat, though this has been shown not to be the case. The form most common is a truncated cone; but hemispheric is not uncommon, and occasionally they may be pedunculated.

"The surface is sometimes smooth, sometimes slightly irregular; rarely it presents a series of small irregularities arranged similarly to those of the skin. Sometimes the surface of the tumor may become covered with fine drops of fluid while it is being examined; these drops come from small glands in the subjacent tissue, and their ducts may be seen by close inspection with the aid of a powerful loupe. The surface usually bears hairs, varying in length with the age of the patient. It is rare to see in children anything but a fine down.

"The site of the tumor is important; commonly they ride the lower outer part of the sclero-corneal junction; more rarely they are purely conjunctival; a completely corneal seat is the least common. In four instances only has the seat been the caruncle. The histology of these tumors shows their close relation to the skin; all the parts of the true skin are to be found; hair glands and sebaceous glands are almost constant; on the other hand, sweat glands are comparatively rare, though not so infrequent as they were held to be by the earlier observers."

Under the name of lipo-dermoids, Pignatari includes those forms which are congenital and have a superficial layer like that of dermoids, but which differ

in containing a large quantity of fat, as well as certain other elements, acinous glands and smooth muscle fibres, which are less frequent in this form. The normal situation of a lipo-dermoid, in which some 70 per cent are found, is between the insertions of the rectus superior and the rectus externus. The structure of these lipo-dermoids varies within wide limits; sometimes the dermal features predominate, sometimes the fat. Often there will be so little development of the cutaneous elements that they may be overlooked, unless series of sections are taken of the whole mass. In a considerable number of cases a mass of cartilage will be found in the deeper part.

The pure conjunctival lipoma is a tumor developed in the subconjunctival tissue and devoid of all cutaneous elements. Many of the cases which appear to be lipomata on clinical examination, prove to be lipodermoid when subjected to the microscope. Great difference of opinion still prevails between pathologists as to the constitution of lipomata, and on their congenital origin.

MIXED TERATOMATA.

As far back as 1888, Gallenga suggested this third division for these tumors, and placed in it those cases in which lipomata were found to contain acinous or acino-tubular glands, or remains of the semilunar cartilage. Later, Gallenga redivided this group into two: (1) mixed teratoma with mucous surface; (2) mixed teratoma with cutaneous surface. The origin of these is probably different from that of the tumors so far considered.

Mixed teratomata contain acino-tubular glands, and these glands often very closely resemble the lacrimal in structure. Gallenga and some others are inclined to consider them really derived from the lacrimal gland, and to be simple ectopia glandis; they reason thus from the fact that the secretion from the gland is neither wanting or abnormal. Gallenga thinks also, that the growth of this aberrant gland accounts in some cases for the presence of cartilage or bone, those fragments having been cut off from the orbital margin by the gland in its extension. Probably, however, in most cases the cartilage is derived from the cartilage in the membrana nictitans, which has become, in the course of development, adherent to the eye.

Concerning the development of teratomata, Pignatari accepts Gallenga's views in accordance with which the typical dermoid is a part of the lid margin which has become adherent to the globe, and has gradually become separated from the lids by the development of abnormal lines of division, which form furrows growing gradually deeper until the dermoid is cut off from the lid and remains a part of the globe. The other form of teratoma is due, in his opinion, to ectopia of some part of the lacrimal gland, which takes up a new position because of the faulty direction of some of its parts. The presence of amniotic bands is only a rare cause.

A comparatively large proportion of dermoids is associated with some faulty development of the eye lids or face. Presumably, coloboma of the lid is chiefly due to the adhesion of a part of the lid-margin to the eye and its separation afterwards; thus a gap is left in the lid, the coloboma, which is accurately filled by the dermoid.

FIBROID TUMORS.

The idea prevailing with practically all authors is that fibroid tumors originate from scar tissue, most of them being simply hyperplasia of this tissue. This is agreed to by Parsons[7], but he says there are cases which are apparently true fibroma. Regarding the structure, he says: "They consist of irregular bands of fibrous tissue, with scant flat and round cells."

CONCLUSIONS.

After reading the literature on corneal tumors and considering this case, I have come to the following conclusions:

(1) Corneal tumors occur less frequently than the literature would indicate.

(2) In order to be classified as a corneal tumor the growth must have no connection with the limbus at any place.

(3) That dermoid may be used as a

term to classify congenital tumors, even those of simple histologic structure containing no dermal tissue, hairs or sweat glands.

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DETERMINATION OF MINIMUM LIGHT SENSE AND RETINAL DARK ADAPTATION WITH PRESENTATION OF A NEW TYPE OF PHOTOMETER.

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This paper reviews the principal hypotheses with reference to light and dark adaptation, and suggests a practical photometer, using a radioactive substance as a standard of comparison. With three illustrations, and experimental findings with this instrument.

The retina is the physiologic point of contact between the external stimulus, light, which the eyes require to convert the world without into a place of form and dimension, luminosity and color; and the final perception of these as "definite qualitative entities in consciousness." The conversion of the physical into a psychologic stimulus is brought about by demonstrable structural, chemic and electric changes engendered by the action of the light waves upon the retinal neuro-epithelial layer of rods and cones.

Reference to one monograph (J. Herbert Parsons, An Introduction to the Study of Color Vision, Cambridge, 1915) is sufficient to show the vast literature dealing with the complex elements, singly and combined, that go to make up this one phase of the visual act. The whole question may be roughly reduced to the one statement that all visual sensations are dependent upon the power that the retina possesses of adapting itself to, and converting into nerve stimuli, light vibrations of varying degrees of intensity.

The simplest example of light and dark adaptation of the retina is the experience of common knowledge, that if we pass from the bright sunlight into a dimly lighted room we cannot plainly distinguish objects until our eyes have become accustomed to this feeble illumination. If, again, we go out into the sunlight we are dazzled by its brightness, until the eyes have once more become adjusted to this degree of light. From the physical standpoint this is entirely a question of the intensity of the external stimulus. From the psycho-physiologic viewpoint, these phenomena are dependent upon certain material alterations which take place in the nerve cells of the retina, in turn exciting neuro-visual consciousness.

It is with this latter aspect that we are concerned, for we may assume that the external stimulus as such exists whether or not we have eyes to see; therefore, we may dismiss from consideration all the qualities of light as vibratory energy in ether, except its quantitative variability, on which depends the two great classes of sensation into which our consciousness of light may be divided, viz.: color sensation and colorless sensation.

Light vibrations are converted into nerve stimuli by the neuro-epithelial rods and cones which make up the outer layer of the retina. It is necessary to remember that there are no rods at the fovea centralis, but passing outward from the

central spot rods begin to make their appearance in increasing numbers, until at the periphery they far outnumber the cones. Parsons distinguishes three areas, as follows: The fovea centralis measuring 0.24 mm. to 0.3 mm. and subtending a visual angle of from 55′ to 70′. A rod free area measuring 0.8 mm. and subtending 3° 3′. The macula measuring from 1 to 3 mm. and subtending 4° to 12°.

Beneath the layer of rods and cones is the layer of pigment epithelium which, tho counted as part of the retina, is in reality outside. The cells of this layer are hexagonal in section and have processes passing up between the rods and cones. The cells contain minute needle shaped crystals of pigment. In the rods alone there is found the substance known as rhodopsin or visual purple, which may only be demonstrated after the eye has been in the dark, as it is completely bleached out by the action of light and is supposed to be reformed only in contact with the pigment cells. In the dark the pigment crystals are found in the cells close to the nucleus, but when the eyes are exposed to light they wander up into the cilia-like processes between the rods and cones. With these anatomic facts in mind let us consider the relationship that can be made out to exist bebetween the varying intensities of the external stimulus light and the resulting sensations elicited.

Let us assume, for example, that we are seated in a room brightly and uniformly illuminated from a source capable of gradual reduction in power until the room is in complete darkness. Under the bright illumination the eyes may be said to be light adapted, or *photopic* (nomenclature of Parsons), we see all objects distinctly and recognize colors. As the illumination decreases in intensity the yellows, normally the brightest colors, are exceeded in brightness by the greens, the blues become lighter and the reds darker. These changes in the relative brightness of colors constitute the well known Purkinje phenomenon. Finally, we can no longer perceive colors at all and objects take on various shades of grey. Later, we can only perceive the form of objects, and then comes complete darkness with no visual sensations. The eyes have been gradually becoming dark adapted or *scotopic*.

Under the conditions described we know that the following changes have been taking place in the perceptive end-organs of vision. In the photopic eye the rods are free of all demonstrable visual purple, and the pigment crystals are found in the cilia between the rods and cones. As dark adaptation proceeds the crystals of pigment migrate back into the cell body, and visual purple becomes evident in the base of each rod. On these anatomic facts there have been built up many theories, two of which must be briefly quoted, (1) The visual-purple theory of Edridge-Green and (2) the duplicity theory of v. Kries.

F. W. Edridge-Green (The Hunterian Lecture on Colour Vision and Colour Blindness, 1912), states his theory as follows: "A ray of light impinging on the retina liberates the visual purple from the rods and a photograph is formed. The rods are concerned only with the formation and distribution of the visual purple, not with the conveyance of the light impulses to the brain. The ends of the cones are stimulated thru the photo-chemical decomposition of the visual purple by light (very probably thru the electricity which is produced); and a visual impulse is set up which is conveyed through the optic nerve fibers to the brain. The character of the stimulus varies according to the wave length of the light causing it. In the impulse itself we have the physiologic basis of the sensation of light, and in the quality of the impulse the physiologic basis of the sensation of color."

Edridge-Green feels that he has proved his theory on evidence which he gives in his book. It is not necessary to go into this except to further quote: "that the visual purple is the essential factor in the retina which enables it to transform light into visual impulses. I find four depressions or canals which lead into the larger depression of the external fovea. These canals appear to have smaller branches and serve to conduct the visual purple into the part of most acute vision. The cones which are present in the fovea

have very long outer segments which would present a greater surface for photo-chemic stimulation. I have examined under the microscope the retinas of two monkeys which had been previously kept in a dark room for forty-eight hours. The yellow spot was the reddish part of the whole retina and the visual purple was seen to be between and not in the cones."

The duplicity theory of v. Kries (Parsons) states that achromatic scotopic vision is carried out thru the medium of the rods alone, the cones being the organs of photopic vision. In dark adaptation with low intensities of light, vision is performed by the rods stimulated by the visual purple, as the intensity of the light increases the liminal stimulation values for the cones is exceeded, and vision is carried out both by the rods and cones (chromatic scotopia). At still higher light intensities vision is carried out chiefly or wholly by the cones (photopia), but whether the rod effects are added to the cone effects, or are abolished, is as yet uncertain.

It will be seen that the formation of the visual purple is, at least, the basis of dark adaptation. In everyday life there is always a moderate amount of scotopia present which is constantly varying to meet the requirements of the moment. We cannot perceive colors unless the light stimulus is above a certain intensity whose liminal value Parsons calls the specific or color threshold. Below this is the colorless interval of light perception, which increases with the amount of dark adaptation present. It is with this interval that my studies are chiefly concerned.

In every physiologic process there is a stimulus which excites, and a resulting reaction, or sensation. There is, of course, a more or less constant relationship between the stimulus and the reaction; and in any investigation it is usual to reduce the external stimulus to its simplest form and then study the resulting sensations, or to observe the simplest reaction that can be elicited by a variety of stimuli. The simplest visual sensation that we can have is the recognition of light, and the simplest stimulus is one that will just produce this reaction under approximately known conditions of retinal adaptation.

It occurred to me that the radioactive preparations so extensively used in the manufacture of luminous watch-dials and other instruments, would furnish an ideal light stimulus of constant value and low intensity, and with the coöperation of the "Cold Light Mfg. Co." of Denver and New York I obtained a series of discs of the radioactive substance which they put out under the trade name of "Marvelite." This substance is sulphid of zinc impregnated by radium: the latter constituent I have proved to my own satisfaction by numerous photographic experiments.

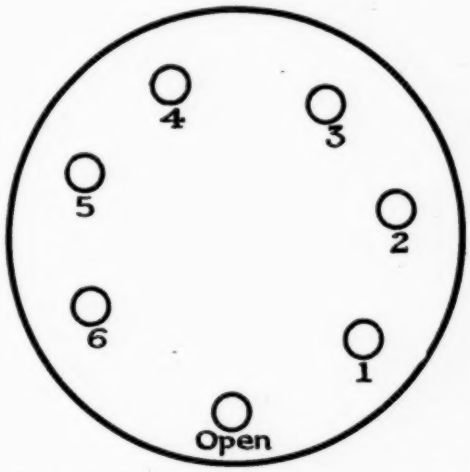

Fig. 1. Disc with openings carrying "Marvelite" exposed or covered with varying thicknesses of celluloid.

The Cold Light Mfg. Co. has instituted photometric measurements concerning the compound, and so far they have been able to determine that there is a rapid initial increase in the luminosity of the radium luminous compounds, followed by a decrease over a period of about six months, the rate of decrease then becomes slower and and slower, until at the end of one year the luminosity attains a value which is sensibly constant. The "Marvelite" discs which I am employing in my ex-

periments have been in my possession for a year.

As my experiments were to be along clinical lines I have calibrated these discs in the following manner. One was chosen as a standard which would just give the sensation of light as soon as I had passed from a well lighted room at midday into a photographic dark room. This disc is considered the minimum light stimulus to the light adapted eye. By photographic exposures I obtained a series of discs of similar intensity and by covering them with layers of No. 5 opaque white celluloid I determined, by experiments carried out at night after two hours in an absolutely dark room, that my minimum lightsense in full dark adaptation was represented by the standard disc covered by five layers of the celluloid.

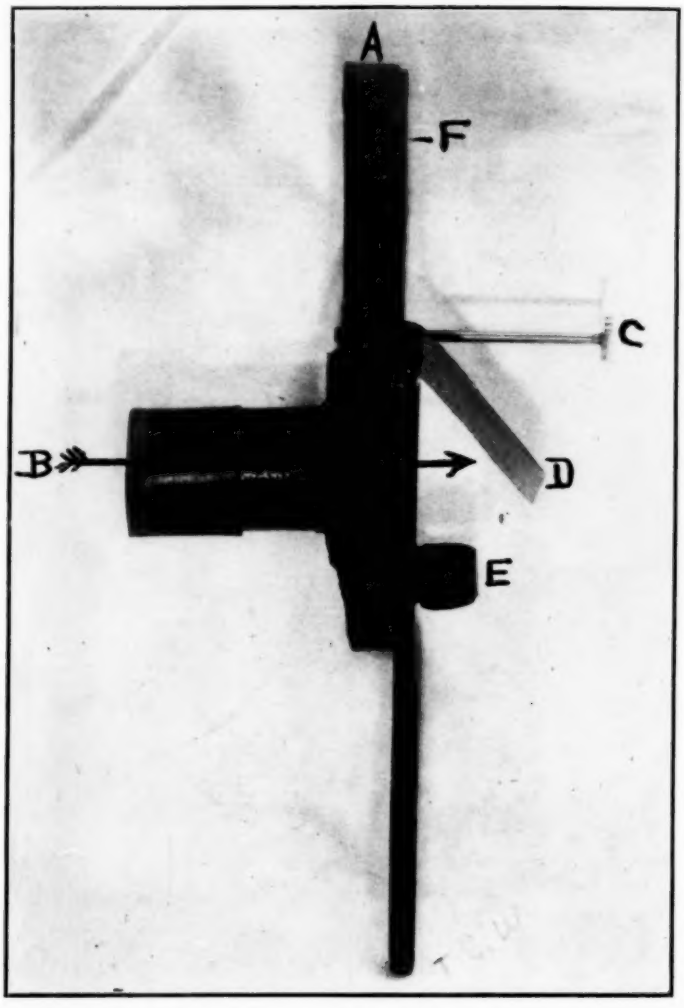

Fig. 2. The photometer described in the text. At F there is an opening through which may be read off the number of the "Marvelite" disc which is before the eye.

Six of these discs were then mounted on a circular plate (Figure 1), Disc No. 1, being the uncovered "Marvelite Button" accepted as the minimum effectual stimulus for the photopic eye. Disc No. 6 covered with five layers of celluloid being the minimum light stimu-

lus under full dark adaptation or scotopia. Discs Nos. 2, 3, 4, 5 and 6 being covered with 1, 2, 3, 4 and 5 layers of the celluloid. I have, therefore, six light stimuli of different degrees of intensity. The following apparatus was then constructed and certain experiments undertaken:

Figure 2 is a photograph of the photometer. The large wheel (Fig. 1) is mounted in the Box A and can be revolved before the eyepiece B by the handle C. When the opening in the wheel is before the eyepiece the observer is, of course, looking straight thru the instrument against the reflector D, which is covered with greenish blue paper to correspond with the faint greenish glow exhibited by the "Marvelite" discs of the higher intensities. The object E on the handle of the instrument is to hold a small electric light for experimental purposes to be later described. The "Marvelite" test discs are 7 mm. in diameter. The instrument is generally used at 10.5 cm. from the eye. The retinal image is therefore 1 mm. and subtends a visual angle of 4°. Figure 3 represents the "blinders" which are used in the application of the test. The slides are easily removed and the photometer brought into position and kept light proof by a leather cuff.

Let us for the moment anticipate the many problems involved and assume that the instrument is of accepted usefulness. The "blinders" are adjusted with the shutters out of the eyepieces. One shutter is replaced and the instrument is adjusted over the other eye-tube. The test object wheel is set at "open" and the observer is told to watch the round spot of light. The "Marvelite" disc No. 1 is now quickly turned into place and the individual should immediately see it. The actual test of the dark adaptation is made as follows. The "blinders" are closed and the patient is asked to keep the eyes shut. At the end of ten minutes the photometer is adjusted over one eye and the individual is asked to say when the light spot first becomes visible. The test-wheel is now slowly revolved so that the "Marvelite" discs of the weak intensities first appear before the eye. The moment the patient says, "now I see light," the number of the disc is noted and the result written in the form of a fraction the numerator of which denotes the patient's mini-

Fig. 3. The "blinders" which are fastened over the eyes.

mum light sense, the denominator the minimum light sense which numerous observations have indicated as the normal. After ten minutes in complete darkness, disc No. 5 should be seen. If the dark adaptation of the retina is normal the test is completed in ten minutes; this statement, I think, can be substantiated by the experimental work now to be described.

EXPERIMENT 1. — Is the Marvelite Disc No. 1 the minimum light stimulus for the light adapted or photopic eye? Under ordinary conditions of everyday room illumination, yes. Under full light adaptation or what may be termed light excess induced by looking at the clear sky on a bright day, no. But even under the latter conditions disc No. 1 becomes evident within thirty seconds after the eye is brought from light into darkness, which proves that the range of light adaptability even in the photopic eye is exceedingly wide. This has been proved by experiments on many individuals. If we accept Edridge-Green's theory, then we must assume, that in the light exhausted eye the visual purple is very rapidly reformed, or that it still preserves photochemic activity even in the bleached out state.

Just how greatly the sensitiveness of the retina to light is increased by dark adaptation is proven by the comparative luminosity of the "Marvelite" disc No. 1 under photopic conditions and in scotopia. In the latter state of the retina the disc is sufficiently intense to illuminate the tube of the eye piece and to slightly raise the minimum light sense. For example, if Disc No. 1 is viewed under full scotopia and the test wheel is then quickly turned to disc No. 6, the latter cannot be distinguished for a few seconds, altho it represented the minimum light sense just a few moments before. Under photopia disc No. 1 can just be discerned as an extremely faint light.

EXPERIMENT 2.—Dark adaptation is very rapid at the beginning of the process and exceedingly slow toward the completion. When the light adapted eye is brought from light into darkness disc No. 2 will become visible in about thirty seconds. Disc No. 3 requires at least ninety seconds of dark adaptation. Disc No. 4 is seen in from three to four minutes and after ten minutes disc No. 5 is just discernable. After ten minutes of dark adaptation the process becomes very slow. In observations made at midday disc No. 5 represented the minimum light stimulus after ten minutes; this was not lowered during an hour's experimentation, altho the luminosity of disc No. 5 became appreciably greater. After two hours observation in a dark room disc No. 6 could just be seen.

As Parsons has said, experiments in dark adaptation are much influenced by the condition of retinal adaptation at the beginning of the observations. I have found that if the individual to be tested has been sitting in a dimly lighted room his photopic minimum light sense may be represented by disc No. 2. With this in view I experimented with a small electric light mounted on the back of the photometer with the idea of bringing all eyes to the same degree of light adaptation before beginning any tests. This has been found unnecessary for the reason that dark adaptation, according to my observations, progresses with definite constancy within all periods under an hour. Disc No. 2 as the minimum photopic light sense only indicates that there is already that amount of dark adaptation present; disc No. 5 may become the minimum light stimulus in less than ten minutes, but beyond that time there will be no appreciable lowering of the light sense, certainly not within the time that one has to devote to a clinical test.

To obtain uniform results it is well to allow the patient to sit in a moderately lighted room for a while before the test is to be made. The photopic minimum light sense is then tested by bringing the instrument before the eye, the patient should immediately see disc No. 1. If there is some slowness in this, beyond sixty seconds, it is indicative of slow dark adaptation. The blinders are then put on and the patient allowed to wait for ten minutes. At the end of this time each eye is tested and

disc No. 5 should be seen. If disc No. 5 is seen the test may be discontinued and the dark adaptation noted as normal. If disc No. 4 represents the minimum light sense after ten minutes the test should be continued for ten minutes longer, when, if disc No. 4 still represents the minimum light sense, the result may be noted 4/5 and dark adaptation considered as "slow." A minimum light sense below disc No. 4 is considered "abnormal."

For the time being I have adopted the following chart to preserve a record of examinations.

		Min. Light Stim.	Dark Adaptation
Immediate	R.	Disc. No.	
	L.	Disc No.	
After 10′	R.	Disc No. /5	
	L.	Disc No. /5	
After 20′	R.	Disc No. /5	
	L.	Disc No. /5	

Remarks:

EXPERIMENT 3.—Dark adaptation occurs in each eye independent of its fellow and is not influenced by light adaptation of the other eye. One eye was kept in darkness for one hour, the other was left open, the experiments being made in a brightly lighted room at midday. Dark adaptation proceeded as heretofore described, disc No. 5 becoming visible in ten minutes and remaining the minimum light stimulus during an hour of further experimentation. Ordinary light stimulus of the other eye had no influence on the dark adaptation of the other eye. Excessive light stimulation of the open eye, e. g., looking directly toward the sun, had no appreciable effect on the dark adapted eye, except that for a moment the dazzling occurring in the light adapted eye seemed to confuse the dark adapted eye. This seemed more the result of the vast difference in the cerebral impressions than any effect on the retina, as the disc could be seen surrounded by a halo of after-dazzling.

EXPERIMENT 4.—Dark adaptation of one eye does not hasten dark adaptation in the other. After one eye had been kept in the dark for one hour, the other eye was closed and frequent observations made. It was found that dark adaptation proceeded in exactly the order before noted, that is to say it took ten minutes to bring the sensibility of the retina down to disc No. 5.

EXPERIMENT 5. — Dark adaptation proceeds equally in both eyes. If both eyes are tested simultaneously it will be found that dark adaptation is the same in each eye after the same period of time. This has been confirmed by numerous tests and, therefore, I feel that it may be stated that dark adaptation, altho an independent process of each eye, is an extremely constant and uniform chemico-physiologic process. If, in clinical determinations, one eye is found to be different from the other, after the same period of time, it is more than suggestive that there is some abnormality in the retinal adaptation.

EXPERIMENT 6.—Is there a summation of effect in the dark adapted eyes? Some observers have found that the minimum light stimulus required is less with binocular vision than with monocular vision. Working with these light stimuli of exceedingly low intensity I have not been able to confirm these findings.

EXPERIMENT 7.—Full dark adaptation is very quickly overcome by exposure of the eye to light. If, after full dark adaptation, the eyes are completely exposed to light, dark adaptation will be overcome in thirty seconds at the most; that is to say, that the minimum light stimulus will be raised from disc No. 5 to disc No. 1, and it will again require the minimum ten minutes to bring the minimum light sense down to disc No. 5. In other words, the bleaching out of the visual purple is very rapidly accomplished if the whole retina is exposed. If, however, only a restricted portion of the retina is exposed to light, the minimum light sense of that portion of the retina is raised, but very quickly regains its minimum light sense. This, to my mind, rather confirms the theory

of Edridge-Green that there is a flow of the visual purple.

Experiments in regard to this were conducted in the following way: Both eyes were kept in the dark for twenty minutes so that the minimum light sense was represented by disc No. 5. Then the blinders were entirely removed and the eyes exposed to bright daylight for thirty seconds; the blinders were then replaced and it was found that the minimum light sense had been raised to disc No. 1. Furthermore, it required the usual ten minutes to again lower the light sense to disc No. 5. After full dark adaptation was again obtained the eye was exposed to a bright light thru a small aperture (5 mm.). This raised the minimum light sense to disc No. 1, but within sixty seconds the minimum light sense was again disc No. 5. In my opinion dark adaptation proceeds regularly over the whole retina. It may be locally bleached but this portion is again quickly sensitized by a flow of the visual purple to the spot, rather than by immediate reformation, otherwise it would require a much longer time to lower the light sense of the restricted area. From these experiments I am inclined to believe that a test of a restricted portion of the retina, such as is made by my photometer, is sufficient to judge the adaptability of the whole retina.

Experiment 8.—Is there any dark adaptation at the central part of the retina? It has already been noted that Parsons distinguishes three areas in this part of the retina, the fovea, a rod-free area and the macula containing a few rods. Used at 10.5 cm. from the eye the 7 mm. test object subtends a visual angle of 4°. This would overlap the rod-free area slightly as this portion is estimated by Parsons to subtend 3° 3′. On the other hand it covers the minimum area allotted to the macula proper of from 4° to 12°. In the discs of low intensity the area of the disc is less than 7 mm. As these discs do not emit any divergent radiations and can only be perceived as light "spots" of very low intensity, irregular in outline and not luminous in the periphery, I calculate their diameter to be about 5 mm. *Undoubtedly there is marked dark adaptation at the macula and if the size of the discs is reduced to 5 mm, dark adaptation can be shown to be present to the same degree in the rod-free area.*

If, however, the discs are viewed thru a pinpoint opening, thus reducing the visual angle practically to zero and making visibility entirely a question of luminosity, disc No. 2, even under complete dark adaptation, becomes the minimum light stimulus. I do not find, however, that the light sense is lower than disc No. 2 even in the extramacular region when tested with the pinpoint disc. I have reached the conclusion, therefore, that there is marked dark adaptation at the macula and in the rod-free area thereof, if the test-object is sufficiently large to subtend a definite angle. At the exact fovea, or in the periphery for that matter, when probably a few cones are stimulated by the point of light, there is very slight dark adaptation below the minimum light sense of the photopic eye. Conclusions in this regard are as yet uncertain. This particular phase of the question is open to much further investigation.

Finally, the work which I present at this time, is to a large degree confirmation of the findings of others; the instrument and the experiments are admittedly elementary.

The manufacturers of "Marvelite," with the coöperation of the United States Bureau of Standards, are making a scientific effort to standardize the luminosity of their product. It is probable that they will be able to produce a radiant substance of uniform value, which can be accurately calibrated. Clinically I have used the test for a year and have found the results extremely interesting and worthy of further study. These reports with further experiments I reserve for a future communication.

COMMON CHANGES IN REGULAR ASTIGMIA AND THEIR CAUSES.

EDWARD JACKSON, M. D.

DENVER.

This paper points out the error of supposing astigmia remains unchanged throughout life. Tabulates the changes found in 1,294 eyes remeasured after five to seventeen years, and the relation of corneal to total astigmia. With one diagram. Read before the Section on Ophthalmology, American Medical Association, June 12, 1918.

The first reported cases of astigmia were of high degree, so that moderate changes in the amount or meridians would not be very noticeable. The first correcting lenses, determined with considerable difficulty, were accepted as an accurate correction thruout life, except in the case of the astronomer Airy,[1] who remeasured his astigmia after nearly 20 years and found it had somewhat diminished. As we would express it today:

His correction of —6.5 ⊂ —5.5 cyl., had changed to —4.5 ⊂ —4.0 cyl.

Donders[2] classed the great mass of cases of astigmia as congenital, recognizing as acquired astigmia that depending on disease, or injury, or incision of the cornea, or upon traumatic displacement of the crystalline lens. He states, "In the great majority of cases it is congenital."[3] This thought was dominant in all his writings on the subject. He implies some skepticism as to the change in Airy's case when he says: "With a man like Airy we may assume that he observed for his farthest point with unaltered accommodation; otherwise we should venture the supposition that, in his earliest observations, in consequence of accommodation on the approach of the point of light, the myopia in the vertical meridian had turned out too great, whereby the recession of the farthest point with the increase of years (which certainly with the existing degree of myopia is extremely rare), should only apparently have taken place."

Knapp's[4] early observations with the ophthalmometer revealed the corneal astigmia unchanged during the brief time over which they were repeated. Javal's[5] statistics dealt with single observations upon each case. The same is true regarding the statistical record of 806 astigmic eyes published by Burnett.[6] The thought of ophthalmologists regarding astigmia was given a strong bent toward the conception that the astigmic condition of an eye was permanent. It was believed to be a congenital characteristic, that each eye carried from the cradle to the grave, unless altered by some accidental circumstance. This was in strong contrast to the earlier writings of Donders and others regarding the increase of hyperopia with age, the progressive tendency of all cases of myopia in the early part of their course, and the senile myopic change that gives second sight.

Early in his work on ametropia, the writer encountered some rather striking cases of progressive hyperopic astigmia, including his own eyes which were repeatedly measured under different cycloplegics. A series of 18 cases of such change was reported to the American Ophthalmological Society in 1890.[7] Attention was also given the changes in astigmia following cataract extraction, and other corneal incisions.[8]

Early in these studies of astigmia, the impression arose that the common belief in its permanence was not well grounded; and the study of this subject was begun by the observation of changes in astigmia presented by patients returning for the remeasurement of their refraction. It was interrupted after a few years by removal from Philadelphia, so that patients previously measured no longer returned for reexamination. These studies, resumed in Denver with a new set of patients, have now been continued for 20 years, and a sufficient number of observations has accumulated to afford a basis for a statistical

study. These include notes on 1,294 eyes of 648 patients who have had their ametropia remeasured after periods of five to seventeen years. All cases of operative or other injury of the eyeball, or of keratoconus or other ocular disease except eye strain, have been excluded. The average period over which the observations of the refraction extended was eight and two-thirds years. Of these eyes 421, or 32.7 per cent, showed less than 0.25D of change in the amount of astigmia present; and 873, or 67.3 per cent, showed at least that much change in their astigmia, as measured for the correction of their ametropia. Of these latter, 394, or 31.2 per cent, showed 0.50D, or more change in the amount of their astigmia.

These cases in which the change was 0.50D. or greater were made the basis of a further statistical study as to the general direction of this change in the astigmia and the ages at which it occurred. The results of such study are embodied in Table I.

The first column gives the ages of the patients thus: Those given as 10 years were under 10 years when first examined; but the large majority were over 10 years at the last examination. Those given as 20 years were between 10 and 20 when first examined, but most of them over 20 when last seen. This plan is followed thruout the table, except that the line for 70 years includes all patients that were over 60 years when first examined.

Column A gives the number of eyes in which the change was an increase of astigmia with the rule, or a decrease of astigmia against the rule, or a passing of astigmia against into astigmia with the rule. Astigmia with the rule was made to include all cases in which the axis of the convex correcting cylinder was placed within 30° of the vertical, or the axis of the concave correcting cylinder within 30° of the horizontal; i. e., plus axes between 60° and 120°, or minus axes between 150° and 30°. Astigmia against the rule includes all cases in which the axis of the convex correcting cylinder lies between 150° and 30°, and the axis of the concave correcting cylinder lies between 60° and 120°.

Column B gives the number of cases in which the change was a decrease of astigmia with the rule, or an increase of astigmia against the rule; or where astigmia with the rule changed to astigmia against the rule as these terms are defined in the preceding paragraph.

Column C gives the number of cases of oblique astigmia in which the change was 0.50D. or more in amount, and in which the direction of the meridians for either convex or concave correcting cylinders lay between 30° and 60° on the one hand, or between 120° and 150° on the other.

TABLE I.

	A		B		C		
Age	Cases,	Per Cent	Cases,	Per Cent	Cases,	Per Cent	Totals
10 years.....	17	55	10	32	4	13	31
20 years.....	30	33	51	55	11	12	92
30 years.....	24	28	55	63	8	9	87
40 years.....	14	16	59	71	11	13	84
50 years.....	13	22	42	71	4	7	59
60 years.....	7	25	19	68	2	7	28
70 years.....	2	15	10	77	1	8	13
	107	27	246	62.5	41	10.5	394

From this table it appears that of this 394 eyes (31.2 per cent of all eyes) that showed a change of 0.50D or more in the amount of their astigmia, a majority, 62.5 per cent showed change toward astigmia against the rule, 27 per cent showed a change toward astigmia with the rule, and 10.5 showed changes in astigmia which remained or became oblique.

When we come to consider the distribution of these changes thru the different periods of life, the percentages show certain unmistakable tendencies. In early life, increase of astigmia with the rule, or the tendency in that direction, predominates, affecting 55 per cent in the first period. But as life advances, this tendency becomes less frequent, until in the last period it is shown in only 15 per cent of eyes. On the other hand, the tendency for astigmia with the rule to diminish, or the tendency toward astigmia against the rule, increases as life goes on. In the first period, it was shown by only 32 per cent. But in the last period, it was manifest by 77 per cent of eyes that remained without recognizable change in amount thruout the period they were under observation. The proportion that remain unchanged is least during the period of growth, but is considerable at all times of life, varying from 23 per cent before 10 years to 39 per cent after 50 years.

It must not be forgotten, however, that this diagram represents only the changes that occur during an average period of eight and two-thirds years. This is only one-fifth of the average life of these patients. It is extremely probable, if not absolutely certain, that many of this 32.7 per cent that showed no change in the amount of astigmia during the period

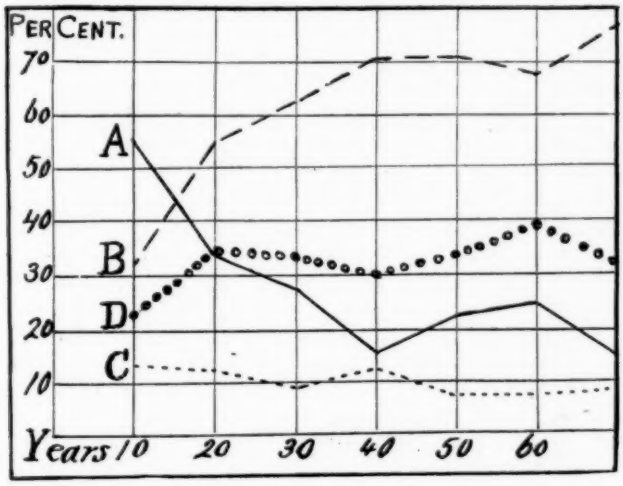

Figure 1.

the eyes examined. On the other hand, the number of cases of astigmia that vary in amount but remain oblique does not vary much (less than 4 per cent from the average of 10.5 per cent) thruout life. These facts are most easily grasped when presented graphically as in Fig. 1.

While these opposing tendencies are unequally represented, or predominate at different ages, it must be noted that each of them is represented at every period of life. There is no period of life at which any change of astigmia may not occur, quite apart from trauma of any kind, or from recognizable ocular disease. In Fig. 1 has been added a fourth line D showing the number of of observation have or will experience such changes at other periods of life.

CAUSES OF CHANGE IN ASTIGMIA.

It is generally assumed that the eye presents three dioptric surfaces bounding its dioptric media. But to the anterior surface of the cornea and anterior and posterior surfaces of the lens must be added the posterior surface of the cornea, and the surfaces bounding the different layers of the crystalline lens. Each layer of the crystalline lens has its individual index of refraction, differing from that of every other layer, so that each layer constitutes a distinct dioptric medium; and every one of

these dioptric surfaces may be astigmic.

It is customary to speak of the total astigmia of the eye as the resultant of its corneal and lenticular astigmia. But Tscherning[9] points out that this is not strictly exact. He calls the astigmia due to the anterior surface of the cornea the ophthalmometric astigmia; and the other possible factors that unite to cause the "subjective" or total astigmia, he groups as together causing what he calls the "supplementary" astigmia. Any difference between ophthalmometric and subjective astigmia is to be counted as supplementary. Let us consider whether these changes in astigmia are due to changes in the ophthalmometric or the supplementary astigmia. This may be detected by comparing the ophthalmometric readings with the total astigmia.

Ophthalmometric readings admit of a certain error of observation that makes differences of 0.25D or 0.50D too small to give reliable results. Those eyes were therefore selected that showed a change of 1D or more in the amount of astigmia for a study that might indicate where the change was located. The successive ophthalmometric readings have all been made with the ophthalmometer of Javal and Schiötz as made by Goubeaux of Paris, model of 1889. As before noted, all cases of distinct keratoconus have been excluded.

The following cases have been selected as being all in which the change of astigmia observed amounted to 1D or more. They illustrate the different relations between the ophthalmometric changes and those noted in the subjective astigmia. In the first group, the changes in anterior corneal surface show some correspondence with the changes in the total astigmia. In a few the change of corneal surface is exactly such as would produce the change in subjective astigmia. In other cases, the correspondence is not so exact, but evidently the anterior surface of the cornea has been one factor in the changes that have altered the astigmia. Under the case number is given the age of the patient when first examined and the age at the last examination. In the second column is given the ophthalmometric astigmia, and in the third column, the subjective astigmia found at the same time. R. and L. indicate the right and left eyes.

GROUP A.

	Ophthalmometric Astigmia	Change	Subjective Test Astigmia	Change
Case 1—				
8	R.+1. cyl. ax. 90°		R.+1. cyl. ax. 83°	
	L.+0.75 cyl. ax. 90°		L.+0.75 cyl. ax. 90°	
17	R.+3. cyl. ax. 75°	+2.	R.+2.75 cyl. ax. 80°	+1.75
	L.+3. cyl. ax. 105°	+2.25	L.+2.75 cyl. ax. 113°	+2.
Case 2—				
12	R.+5. cyl. ax. 105°		R.+4. cyl. ax. 105°	
	L.+4. cyl. ax. 75°		L.+4. cyl. ax. 77°	
20	R.+5.75 cyl. ax. 110°	+0.75	R.+5.50 cyl. ax. 110°	+1.50
	L.+4. cyl. ax. 85°	0.	L.+4.50 cyl. ax. 83°	+0.50
Case 3—				
9	R.+1.25 cyl. ax. 75°		R.+1.25 cyl. ax. 60°	
	L.+1. cyl. ax. 100°		L.+1.25 cyl. ax. 93°	
19	R.+0.25 cyl. ax. 90°	—1.	R. no cyl.	—1.25
	L.+0.75 cyl. ax. 120°	—0.25	L.+0.37 cyl. ax. 150°	—0.87
Case 4—				
14	R.+3. cyl. ax. 65°		R.+4. cyl. ax. 60°	
	L.+3. cyl. ax. 115°		L.+3.50 cyl. ax. 130°	
23	R.+2.75 cyl. ax. 60°	—0.25	R.+3.50 cyl. ax. 55°	—0.50
	L.+1.50 cyl. ax. 105°	—1.50	L.+2. cyl. ax. 115°	—1.50

	Ophthalmometric Astigmia	Change	Subjective Test Astigmia	Change
Case 5—				
16	R.+6. cyl. ax. 135°		R.+5. cyl. ax. 165°	
	L.+0.50 cyl. ax. 90°		L. no cyl.	
21	R.+7. cyl. ax. 145°	+1.	R.+6.50 cyl. ax. 1°	+1.50
	L.+1.50 cyl. ax. 30°	+1.	L.+2.50 cyl. ax. 155°	+2.50
Case 6—				
19	R+1.50 cyl. ax. 100°		R.+1.25 cyl. ax. 95°	
	L.+1.75 cyl. ax. 90°		L.+1.75 cyl. ax. 90°	
31	R.+2. cyl. ax. 100°	+0.50	R.+2.25 cyl. ax. 93°	+1.
	L.+2.25 cyl. ax. 85°	+0.50	L.+2.25 cyl. ax. 90°	+0.50
Case 7—				
22	R.+4.50 cyl. ax. 145°		R.+3. cyl. ax. 7°	
	L.+4. cyl. ax. 35°		L.+3.50 cyl. ax. 10°	
28	R.+6. cyl. ax. 140°	+1.50	R.+5.50 cyl. ax. 5°	+2.50
	L.+7. cyl. ax. 45°	+3.	L.+5.50 cyl. ax. 5°	+2.
Case 8—				
23	R.+2.25 cyl. ax. 100°		R.+1.75 cyl. ax. 90°	
	L.+2. cyl. ax. 85°		L.+1.50 cyl. ax. 95°	
29	R.+3.25 cyl. ax. 90°	+1.	R.+2.50 cyl. ax. 88°	+0.75
	L.+3. cyl. ax. 90°	+1.	L.+2.50 cyl. ax. 103°	+1.
Case 9—				
23	R.+1.25 cyl. ax. 90°		R.+0.37 cyl. ax. 105°	
	L.+1. cyl. ax. 100°		L.+0.50 cyl. ax. 40°	
32	R.+2.75 cyl. ax. 90°	+1.50	R.+1.37 cyl. ax. 82°	+1.
	L.+2. cyl. ax. 110°	+1.	L.+0.75 cyl. ax. 125°	+1.25
Case 10—				
28	R.+3.25 cyl. ax. 105°		R.+2.50 cyl. ax. 105°	
	L.+4. cyl. ax. 90°		L.+2.50 cyl. ax. 88°	
44	R.+4.25 cyl. ax. 97°	+1.	R.+3. cyl. ax. 105°	+0.50
	L.+4.75 cyl. ax. 85°	+0.75	L.+3.75 cyl. ax. 85°	+1.25
Case 11—				
40	R.+4. cyl. ax. 90°		R.+3.50 cyl. ax. 85°	
	L.+4. cyl. ax. 90°		L.+3.50 cyl. ax. 95°	
46	R.+4.25 cyl. ax. 90°	+0.25	R.+3.75 cyl. ax. 84°	+0.25
	L.+4.50 cyl. ax. 95°	+0.50	L.+4.50 cyl. ax. 96°	+1.
Case 12—				
43	R. no cyl.		R.+0.25 cyl. ax. 145°	
	L.—0.50 cyl. ax. 180°		L.+0.50 cyl. ax. 180°	
50	R.+0.50 cyl. ax. 135°	+0.50	R.+0.50 cyl. ax. 115°	+0.50
	L.+1. cyl. ax. 15°	+0.50	L.+1.50 cyl. ax. 165°	+1.
Case 13—				
53	R. Less than 0.25		R.+0.25 cyl. ax. 65°	
	L. Less than 0.25		L.+0.62 cyl. ax. 90°	
66	R.+1.50 cyl. ax. 25°	+1.50	R.+2. cyl. ax. 10°	+1.75
	L.+1. cyl. ax. 175°	+1.	L.+1.25 cyl. ax. 175°	+0.62
Case 14—				
57	R.+0.50 cyl. ax. 90°		R. no cyl.	
	L.+0.50 cyl. ax. 90°		L. no cyl.	
71	R.+0.25 cyl. ax. 180°	+0.75	R.+1. cyl. ax. 175°	+1.
	L.+0.25 cyl. ax. 180°	+0.75	L.+1. cyl. ax. 175°	+1.

The cases in the following group show wide discrepancies between the changes in the ophthalmometric readings and the changes in total or subjec-

tive astigmia. In some cases, the two changes have been in opposite directions, so that the change in astigmia is clearly due to some factor other than the change in the anterior corneal surface.

GROUP B.

	Ophthalmometric Astigmia	Change	Subjective Test Astigmia	Change
Case 15—				
7	R.+0.50 cyl. ax. 90°		R.+0.50 cyl. ax. 90°	
	L.+0.50 cyl. ax. 90°		L.+0.50 cyl. ax. 90°	
18	R.+1. cyl. ax. 105°	+0.50	R.+0.75 cyl. ax. 25°	+1.25
	L.+1.25 cyl. ax. 90°	+0.75	L.+0.75 cyl. ax. 113°	+0.25
Case 16—				
13	R.+1. cyl. ax. 85°		R.+0.62 cyl. ax. 95°	
	L.+1.25 cyl. ax. 90°		L.+0.62 cyl. ax. 85°	
24	R.+1.25 cyl. ax. 90°	+0.25	R.+0.37 cyl. ax. 170°	—1.
	L.+1. cyl. ax. 90°	—0.25	L.+0.37 cyl. ax. 10°	—1.
Case 17—				
23	R.+1.75 cyl. rx. 90°		R.+1.50 cyl. ax. 103°	
	L.+1.25 cyl. ax. 90°		L.+1. cyl. ax. 85°	
36	R.+3.50 cyl. ax. 90°	+1.75	R.+2. cyl. ax. 102°	+0.50
	L.+3.25 cyl. ax. 90°	+1.75	L.+2. cyl. ax. 90°	+1.
Case 18—				
26	R.+6. cyl. ax. 98°		R.+6. cyl. ax. 97°	
	L.+5. cyl. ax. 85°		L.+6. cyl. ax. 83°	
38	R.+6. cyl. ax. 100°	0.	R.+7. cyl. ax. 92°	+1.
	L.+5.5 cyl. ax. 80°	+0.50	L.+6.50 cyl. ax. 83°	+0.50
Case 19—				
28	R.+1.25 cyl. ax. 180°		R.+1.25 cyl. ax. 10°	
	L.+0.50 cyl. ax. 180°		L.+1.25 cyl. ax. 172°	
39	R.+1. cyl. ax. 10°	—0.25	R.+2. cyl. ax. 10°	+0.75
	L.+2. cyl. ax. 125°	+2.50	L.+2.50 cyl. ax. 138°	+1.25
Case 20—				
39	R.+3. cyl. ax. 90°		R.+2.75 cyl. ax. 85°	
	L.+2. cyl. ax. 90°		L.+1.75 cyl. ax. 95°	
51	R.+3. cyl. ax. 75°	0.	R.+2. cyl. ax. 80°	—0.75
	L.+2.50 cyl. ax. 105°	+0.50	L.+0.75 cyl. ax. 85°	—1.
Case 21—				
40	R.+0.50 cyl. ax. 90°		R.+0.75 cyl. ax. 140°	
	L.+0.25 cyl. ax. 90°		L.+0.75 cyl. ax. 30°	
46	R. no cyl.	—0.50	R.+1.25 cyl. ax. 170°	+0.50
	L. no cyl.	—0.25	L.+1.75 cyl. ax. 175°	+1.
Case 22—				
50	R.+1.50 cyl. ax. 90°		R.+1. cyl. ax. 105°	
	L.+2. cyl. ax. 90°		L.+1.50 cyl. ax. 90°	
59	R.+1. cyl. ax. 90°	—0.50	R. no cyl.	—1.
	L.+1. cyl. ax. 90°	—1.	L. no cyl.	—1.50
Case 23—				
55	R.+0.25 cyl. ax. 180°		R.+2.50 cyl. ax. 105°	
	L. no cyl.		L.+2.50 cyl. ax. 88°	
67	R.+0.25 cyl. ax. 180°	0.	R.+3. cyl. ax. 105°	+0.50
	L.+0.25 cyl. ax. 180°	+0.25	L.+3.75 cyl. ax. 85°	+1.25
Case 24—				
65	R.+1. cyl. ax. 75°		R.+0.75 cyl. ax. 40°	
	L. no cyl.		L.+1. cyl. ax. 170°	
73	R.+1.25 cyl. ax. 35°	+0.25	R.+2.50 cyl. ax. 20°	+1.75
	L.+0.75 cyl. ax. 135°	+0.75	L.+250 cyl. ax. 170°	+1.50

	Ophthalmometric Astigmia	Change	Subjective Test Astigmia	Change
Case 25—				
67	R.+0.50 cyl. ax. 30°		R.+0.75 cyl. ax. 15°	
	L.+0.50 cyl. ax. 150°		L.+1.25 cyl. ax. 170°	
74	R.+1.50 cyl. ax. 15°	0.	R.+4.50 cyl. ax. 7°	+3.25
	L.+0.75 cyl. ax. 150°	+0.25	L.+1.50 cyl. ax. 170°	+0.25

In the third group are included cases in which the changes in one eye would place the case in the first group, but the changes in the other eye are such as have occurred in cases of the second group. They emphasize the fact that even in the same patient, at the same time, differing factors may be working to produce changes of astigmia.

GROUP C.

	Ophthalmometric Astigmia	Change	Subjective Test Astigmia	Change
Case 26—				
11	R.+0.75 cyl. ax. 90°		R. no cyl.	
	L.+0.75 cyl. ax. 90°		L. no cyl.	
17	R.+0.75 cyl. ax. 90°	0.	R.+1.50 cyl. ax. 115°	+1.50
	L.+0.75 cyl. ax. 90°	0.	L. no cyl.	
Case 27—				
15	R.+2. cyl. ax. 90°		R.+2. cyl. ax. 85°	
	L.+2. cyl. ax. 90°		L.+2.25 cyl. ax. 100°	
22	R.+2. cyl. ax. 90°	0.	R.+1.75 cyl. ax. 90°	−0.25
	L.+2. cyl. ax. 90°	0.	L.+1.25 cyl. ax. 87°	−1.
	Ophthalmometric Astigmia	Change	Subjective Test Astigmia	Change
Case 28—				
17	R.+1.50 cyl. ax. 90°		R.+0.62 cyl. ax. 90°	
	L.+1.50 cyl. ax. 90°		L.+0.62 cyl. ax. 85°	
25	R.+5. cyl. ax. 100°	+3.50	R.+2.25 cyl. ax. 130°	+1.62
	L.+1.25 cyl. ax. 90°	+0.25	L.+0.37 cyl. ax. 165°	−0.25
Case 29—				
32	R. no cyl.		R. no cyl.	
	L.+0.75 cyl. ax. 15°		L.+1.50 cyl. ax. 5°	
39	R.+1. cyl. ax. 90°	+1.	R.+0.50 cyl. ax. 72°	+0.50
	L.+0.25 cyl. ax. 180°	−0.50	L.+2.50 cyl. ax. 180°	+1.
Case 30—				
35	R.+2.50 cyl. ax. 100°		R.+1.50 cyl. ax. 120°	
	L.+1.50 cyl. ax. 90°		L.+0.50 cyl. ax. 18°	
41	R.+1.50 cyl. ax. 105°	−1.	R.+0.50 cyl. ax. 138°	−1.
	L.+1. cyl. ax. 80°	−0.50	L.+1. cyl. ax. 80°	+1.50
Case 31—				
56	R.+5. cyl. ax. 125°		R.+2.50 cyl. ax. 145°	
	L.+1. cyl. ax. 90°		L.+0.50 cyl. ax. 90°	
68	R.+5. cyl. ax. 115°	0.	R.+3.75 cyl. ax. 102°	+1.25
	L.+0.50 cyl. ax. 105°	−0.50	L. no cyl.	−0.50

In examining Group A, it will be noticed that among the 28 eyes included, 24 showed the change to be one of increase of the astigmia; and that 15 showed increase of astigmia with the rule and 9 against the rule. The 4 in which the astigmia diminished were all cases in which it was changing from with to against the rule. These general tendencies hold good regarding lower degrees of astigmia, in which, however, there is a greater chance of error. So far as these statistics indicate, the changes in the anterior surface of the cornea generally tend in the direction of relative increase of curvature in the vertical meridian, altho to this rule there are exceptions.

Examination of Group B shows that where the change in total astigmia depends chiefly on a change of the supplementary astigmia, the corneal curvatures not showing any corresponding change, the tendency is generally toward astigmia against the rule, this being the case in 16 of the 22 eyes given in that group.

With regard to Group C, which includes 6 cases, we find that in the eyes in which the total corresponded to the corneal astigmia, 3 are with the rule, and 1 against. While in the eyes in which the change of corneal and total astigmia did not correspond, 3 are with the rule, 3 against; and 2 changing from with the rule to oblique, which has the same significance as changing from with the rule to against the rule.

In all three groups, therefore, it appears that the tendency is for astigmia to increase with the rule by increase of the corneal astigmia; and to increase against the rule by increase of the supplementary astigmia.

As to congenital astigmia, there is a sense in which a large proportion of cases may be regarded as congenital in origin. The work of Steiger[10] upon the heredity of ametropia has demonstrated what many observers had believed, that a tendency to astigmia exists in many families. Spengler[11] reported such a tendency in the family of Javal. Out of such a congenital tendency undoubtedly a large proportion of cases of astigmia develop, just as a congenital tendency may determine that in certain persons the hair will get white at an exceptionally early age, or that glaucoma or cataract will develop.

But many of these cases of astigmia of congenital origin differ entirely in their history from such congenital defects, as microphthalmos, or coloboma of the iris, or congenital color blindness. It is certain that in some of the cases the astigmia, although due to a congenital tendency, develops during life. The large proportion of children in whom the astigmia changes after they reach an age when it comes to be accurately measured, and the proportion of these in which the subjective astigmia corresponds to the form of the anterior corneal surface, indicates that many cases of "congenital" astigmia arise during the period of growth. Nor can it be claimed that the corneal development is the only factor in producing these cases of astigmia. In some of them an asymmetric growth of the lens is the most logical inference.

The progressive increase in the number of cases of astigmia changing from "with the rule" to "against the rule" is perhaps the most striking fact brought out. This is not at variance with the recorded experience of others. The statistics of Nordenson[12], which showed such a large percentage of corneal astigmia with the rule were based upon the examination of school children. It was about the time of their publication that the great preponderance of astigmia with the rule was generally accepted. Knapp's[13] original measurements of the cornea found among nine cases one in which a flatter meridian was vertical, and four in which it was more or less oblique. Donders'[14] fifteen cases show one case in which the meridian of greatest refraction was at 2°, another at which it was at 17°, and one at 172°; and in which all but five were the eyes of persons under thirty years of age.

In 1891, de Schweinitz[15] reported one hundred cases of astigmia contrary to the rule. Of his patients, 30 per cent were under forty and 70 per cent over forty. In 1903, Bennett and Clemesha[16] published a study of astigmia, in which they gave the proportions of the different forms of astigmia found at different ages. They found that among persons in the first decade of life, 88 per cent showed astigmia with the rule, while after seventy less than 30 per cent showed astigmia with the rule, and 63 per cent showed astigmia against the rule.

In this paper nothing has been said of changes in the meridians of astigmia, except regarding the three general classes of "with the rule," "against the rule," and "oblique." In Case 16 the change in total astigmia was only 0.25 D, but the change of 75° in the meridians of each eye made it a re-

versal of astigmia, equal in significance to a change of 1D in the amount. But changes in the meridians may be so readily produced by slight changes in one of the factors entering into the total astigmia that a study of changes in the direction of the axis of the correcting cylinder measured in degrees would be more confusing than helpful. An examination of these changes confirms observations based on the amount of rough change of axis; but shows that astigmia is even more variable than the facts here brought out would seem to indicate.

CONCLUSIONS: The facts before us demonstrate that astigmia is liable to change at any period of life. In childhood, it frequently increases because of the increased asymmetry of the anterior surface of the cornea. Such change is usually in the direction of relatively greater curvature in the vertical meridian. But this change may occur at any time of life. In childhood, also, the astigmia sometimes changes because of change in the other dioptric surfaces of the eye; and changes due to this cause become more and more frequent as time goes on. Such changes cause "astigmatism with the rule" to become the exception in the latter part of life, its opposite being now the rule.

This fact makes these generally understood terms entirely inappropriate, and they should be replaced by others, such as: *Direct,* with the meridian of greatest curvature approximately vertical; *inverse,* with the meridian of greatest curvature approximately horizontal, and *oblique* in the same sense in which it is used in this article.

Astigmia is congenital chiefly in the sense that a tendency to asymmetric refraction exists, which works itself out at some stage in the development of the individual.

The variety of the changes that may occur in astigmia point to the interplay of a number of factors that are yet to be studied. The fact that a large majority of eyes undergo such changes to an extent that makes them of practical importance makes it imperative that the astigmia shall be remeasured whenever symptoms arise that may be due to uncorrected astigmia.

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HYPERMETROPIA RESPONSIBLE FOR HETEROPHORIA ASTIGMATISM AND MYOPIA.

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NEW YORK.

This paper contains an explanation and argument based upon statistics of the relative frequency of these different conditions at different ages. It was read before the American Ophthalmological Society, July 10th, 1918.

In the year 1888 the Stevens theories and claims of cures of certain ailments by partial tenotomies caused so much interest that a committee of ophthalmologists and neurologists was appointed to investigate them. In the writer's belief, the results of the new treatment were not satisfactory, but it was adopted by a large number of ophthalmologists all over the country. They were divided into the following six groups as to cause of deviation: muscles too strong, too weak, too long, too short, attached too near the cornea, and malformation of the orbit. Stevens, in his book on Functional Nervous Diseases, states that the superior rectus of one eye is too strong, or the inferior rectus of its fellow is too strong. In either case he calls the vertical tendency "hyperphoria," and cuts the superior rectus of the eye that is on the higher plane. If that fails to correct the tendency, he cuts the inferior rectus of the fellow-eye.

During the investigation by the committee the writer had the good fortune to measure the tendency before and after these operations. During the first period of the operations the writer discovered that the eye operated on was the fixing eye, and, of course, was the eye on the horizontal plane, while its fellow was below it. A commonsense view afforded a simple explanation. Instinctively, man or animal fixes with the better eye, whether or not the fellow-eye has a tendency to deviate. The writer saw cases of tenotomy of superior rectus in eyes with vision 20/20 to 20/15, while the vision of their fellows was 20/30 or 20/40 or even less, this faulty vision being due to a greater error of refraction. Thoroly convinced that the vertical tendency to deviate was due to the difference in the error of refraction, and that the tendency was downward, the writer worked out the causes and discovered that the fault was not in the muscles but in errors of refraction. Those muscles act instinctively to aid the fixing eye by a greater muscular effort, just as an eye with distorting vision produced by lesion causes the eye instinctively to turn. If the fixing eye is better than the two combined, the affected one is discarded.

On this subject a paper was written and read before the ophthalmic section of the Academy of Medicine in 1893, with a report of 200 cases showing the following:

Refraction the same:			
Esophoria	70		(90.1 per cent.)
Exophoria	21		
	—	91	
Hypophoria	1		
Hypoesophoria	5		(9.9 per cent.)
Hypoexophoria	4		
	—	10	
		101	
Refraction different:			
Horizontal tendency	19		(19.2 per cent.)
Vertical tendency..	80		(80.8 per cent.)
	99		
Tendency downward	65		(81.25 per cent.)
Tendency upward..	15		(18.75 per cent.)
	80		

TORSION.

After reading Dr. Savage's paper on Insufficiency of the Oblique Muscles, the writer began to look for cases showing those defects, and found only three, all of which were in eyes having a different error of refraction. This led the writer to prepare and read a second paper before the ophthalmic section in 1898. In this collection of

514 cases the following conditions appeared:

Refraction the same:		
Horizontal tendency....	194	(81 per cent.)
Vertical tendency.....	45	(18 per cent.)
	239	
Refraction different:		
Horizontal tendency....	42	(14.55 per cent.)
Vertical tendency.....	233	(85.45 per cent.)
	275	
Hyperphoria	74	(31 + per cent.)
Hypophoria	159	(68 + per cent.)
	233	

Of these two papers combined, 340 showed the same error of refraction, the tendency to deviate being confined to the horizontal plane, 83.82 per cent., and those showing a vertical tendency, 16.18 per cent.

The remaining 374 cases were anisometropic, showing a horizontal tendency to deviate of 16.32 per cent., and a vertical tendency of 83.68 per cent.

It thus appears that the totals in the two classes of cases are virtually reversed, the percentage being about 84 and 16 per cent. respectively in each class, the difference being about one-seventh of 1 per cent.

The third paper on this subject was published in 1904, in the Archives of Ophthalmology, vol. xxxiii, with a report of only a dozen cases.

EMMETROPIA.

The writer believes that all the external ocular muscles supplied by the third nerve act with the ciliary muscle in accommodation. The four recti muscles are so attached to the globe of the eye that, acting singly, they turn in the direction of that muscle. Any two muscles acting together turn the eyeball in the diagonal of these directions, as in looking around a room. The oblique muscles rotate the eyeball on its anteroposterior axis, keeping the eye in its vertical plane.

The internus and inferior rectus are governed by a common tendon,—the ligament of Zinn,—which is attached around the circumference of the optic foramen except at its upper and outer part. These two muscles are larger and stronger than any other two muscles combined, the internus being the larger and stronger. In accommodation they act together, of course, downward and inward. The superior recti are the thinnest and narrowest of the four recti muscles which pull the eye upward and inward. The inferior obliques, thin, narrow muscles, pull the eye upward and outward, and both are supplied by the third nerve. If the inferior oblique turns the eye outward above to keep it on its vertical plane in accommodation, what action could the superior rectus take except to relax? It is hardly logical to assume that it is the only idle muscle in accommodation supplied by the third nerve. It must be included in the process of accommodation, the superior rectus and inferior obliques counteracting or counterbalancing each other, and holding the upper part of the eye in its original position, while the internus and inferior rectus turn it inward and downward. At the same time the externus and superior oblique relax, so as to place the lower part of the eye directly under the upper part, thus keeping the eye in its vertical plane, turning downward and inward.

When hypermetropia is the same in both eyes, and the vision is normal, the tendency to converge is equal in the two eyes. If esotropia develops, one eye turns downward and inward by increased action of the two muscles to avoid diplopia. We should call this a voluntary contraction, because it is independent of the amount of stress necessary to work with the fellow-eye. That this is independent of the action necessary for accommodation is shown by the fact that there is usually no latent hypermetropia. That is, the eye would accept a +4 D., while the fixing eye would probably have a manifest of not more than 1 D. When under atropin they would both accept +4 D. When they work together (absence of squint), the manifest varies from nothing to probably the full amount of error. When the hypermetropia is different, we have either hypophoria or hypoesophoria, more often the latter. The hypophoria is in the more hypermetropic eye, while the fixing eye is on the horizontal plane.

When hypermetropia differs in the two eyes, and each accommodates to correct its error, the eye with the greater error turns more inward, as it turns more downward and may be thrown on a different plane. Torsion is more apt to be found in myopic eyes differing in refraction. As they cannot work together, the discarded eye is turned outward and downward by the contraction of the externus and superior oblique.

PSEUDO- AND TRUE HETEROPHORIA.

Dr. Savage claims that pseudoheterophoria is the amount that glasses can correct; that true heterophoria is the uncorrected esophoria in hypermetropia, the uncorrected exophoria in myopia, exophoria in hypermetropia, and esophoria in myopia. If it can be shown that what Dr. Savage calls "true" is pseudoheterophoria, something worth while has been gained. Our literature on the subject of accommodation is with emmetropic eyes or eyes made emmetropic by correcting glasses. This is preliminary to what we wish to prove. We will take 13 inches as a comfortable distance for an emmetropic eye. The first impulse is to see distinctly, causing the crystalline lens to increase 3 D. The second impulse is to avoid diplopia by converging to a point at 13 inches. So long as nature provides an emmetropic eye to go with accommodation and convergence to a given point, all goes well. But when eyes hyperopic 3 D. have to accommodate 6 D., an attempt to converge to a point 13 inches away, when the natural convergence would be six and a half inches, all goes wrong. This convergence to 6½ inches is prevented by a volunteer contraction of the externi and superior obliques—the counteraction muscles; unless it is possible for the interni and inferior recti to contract, only half the amount of the ciliary muscle. In such cases, if we do not get a convergent squint, we have either esophoria or exophoria. The cases of exophoria in hypermetropia can be attributed to two causes, working together or separately:

1. The strain or fatigue of the ciliary muscle may come first, which would be indicated by a blurring or, as patients often say, everything turns black before their eyes. This would be followed by a relaxation of the internal and inferior recti muscles.

2. The fatigue in convergence is more likely to come first, as they are antagonized by two opposing sets of muscles, the externi and the superior obliques, to offset the extra physiologic action of the interni in accommodation to a given point, indicated by the extra increased curvature of the crystalline lenses. The indication of the interni fatigue is either a separation of the letters into two sets of letters, or a tendency to do so, indicated by the broadening out of each letter, making the letters unrecognizable. This condition is described by the patient as "the letters dancing all over the page." This may mean either exophoria or esophoria, or alternating struggles against the ciliary muscles to hold convergence at a distance of 3 D. We may have exophoria for the near and esophoria for the distance, which is not at all uncommon, and in other cases exophoria for both far and near, which is often found.

When the hypermetropia is different, the eye with the greater error moves inward and downward, on account of greater muscular effort to correct the difference. Thus hypoesophoria 80 per cent. The hyperesophoria of 20 per cent. is accounted for by the internus and inferior rectus becoming tired, for the same reason that we have exophoria in hypermetropic eyes the same.

If the full correction for the hypermetropia is made, Dr. Savage's true heterophoria turns out to be the pseudoheterophoria. When this full correction is given, the interni and ciliary muscles act in harmony; and neither the exophoria or hypoexophoria any longer exists either near or at the distance. Recently, considerable attention has been given to ciliary muscle fatigue by some of our distinguished ophthalmologists. The most recent was by the chairman of a committee of the ophthalmic section of the American Medical Association for eye

muscle research. This report on the ciliary muscle gave the measurement of fatigue at different ages.

While such reports are interesting, their practical value is questionable. If all errors of refraction are corrected, ciliary muscle fatigue will be confined practically to emmetropic presbyopic eyes, and to those of younger people who hold their work too near the eyes. It is the opinion of some ophthalmologists that amblyopia, in hypermetropia, precedes the squint, and of others that amblyopia comes subsequent to the squint from the non-use of the eye. As this paper is confined strictly to muscles and refraction, no other amblyopic cases are considered.

In the writer's opinion, excessive accommodation causes double vision, followed by alternating squint, which later on becomes a permanent squint of one eye. While the writer agrees with the latter group that the amblyopia is subsequent to the squint, he does not believe that it is directly due to the squint. We have all seen eyes where the squint had existed for years with normal vision in the squinting eye, with or without a glass. The reason for this is that the accommodation in that eye has continued, and all rays of light have fallen upon the retina, including the macula, with the same sharp focus as that of the fellow-eye. This continued sharp focus keeps the retina active, notwithstanding images are ignored or suppressed. When we find a squint of long standing with amblyopia, we find the hypermetropia to be 4 D. or over, with absolutely no accommodation. This produces the amblyopia, because the rays of light fall upon the retina and macula under circles of diffusion. With no focus to stimulate the retina, the eye becomes amblyopic. This eye will show under the test the total error, while its fellow will show about one-fourth. If the two eyes are placed under atropin, we shall probably find that the fixing eye has the same amount of error, with no increase in the refraction of the squinting eye. We have also noticed that very high degrees of hypermetropia, such as 6 or 7 D., do not have squint. Such cases we shall find are amblyopic unless glasses have been worn to correct the error.

ESOPHORIA IN MYOPIA.

Esophoria in myopia is not very common and is found when myopia is the same in both eyes. In these cases we have no accommodation or less than the amount required to stimulate convergence to the point of fixation. The interni take on an independent contraction to produce sufficient convergence to prevent crossed diplopia. A forced action of an individual muscle outside the laws of accommodation and associated movements of the eyes may produce heterophoria, and in the case of myopia produce esophoria. The myopia corrected will force the eyes to accommodate, as in emmetropia, and the convergence produced by the action of the ciliary muscle will correct the esophoria. When the eyes cease to maintain binocular single vision, for the want of a stimulating influence of the ciliary muscle, one eye begets a tendency to diverge and soon becomes more near-sighted. It then begins to turn outward and downward. When the prism test is made to determine the amount of deviation, the eye will move further outward or downward to avoid fusing with the other light. When the externus and superior oblique can no longer overcome the prisms, they give up, and the larger light crosses to the other side and may move above or below and back again to the right or left. It seems that any of the six muscles are liable to come into play to avoid fusing. When these muscles become exhausted in their efforts and the lights are finally brought together, the eyes see a smaller and a more defined light in the larger and more blurred light. Unless the vision is very faulty under the correction, the eyes will work together when the image is made the same size on the retina, even in cases of actual divergence, and even after partial tenotomies, when the muscles are not displaced by the cutting.

While the cases above reported show about 84 per cent. horizontal tendency, when the refraction is the same, and

about 84 per cent. vertical tendency in anisometropia, about 20 per cent. of the 84 per cent. are hyperphoria and 80 per cent. hypophoria. The cases where refraction is the same show 20 per cent. exophoria in hypermetropia and about 20 per cent. esophoria in myopia; so the exceptions to the rule are about the same.

REGULAR ASTIGMATISM.

During the year 1892 the writer adopted the theory that the greater curvature of the cornea is in the horizontal instead of the vertical axis in more than 50 per cent. of the cases after the age of fifty years.

A paper was subsequently prepared and read at the ophthalmic section of the New York Academy of Medicine in 1894. The following is a summary of the 1071 cases therein referred to, grouping those, according to age, in the horizontal axis. Age ten up, 27 per cent.; twenty up, 29 per cent.; thirty up, 34.4 per cent.; forty up, 47 per cent.; forty-five up, 49.3 per cent.; fifty up, 54.4 per cent.; sixty up, 64.3 per cent.; seventy up, 100 per cent.

It was observed that oblique axes increased with age below axes 45 and 135, but those cases were not included. No case of astigmatism was found in children under six years of age, and only about a dozen in children under ten. These were excluded.

At the beginning of the writer's studies the text-books stated that astigmatism was either congenital or acquired. When the theory that the axis in the greater curvature of the cornea changed with age was adopted, a cognate idea was presented; it was observed that in the majority of cases the defect was acquired and that the cases of a high degree were congenital and probably the only ones.

Among the thousands of cases examined, from which the 1071 were selected, no cases of congenital origin were discovered. Still the congenital theory was so widely held among eminent specialists that the writer hesitated to publish his convictions. But an added experience of a quarter of a century has dissolved all doubt. Ophthalmologists agree that, as a rule, babies at birth are hypermetropic; and, as they grow, the eyes become more nearly spheric until the majority become round or emmetropic. Add to this the fact that no cases of astigmatism are found under the age of six in the former list of 1071 cases, or the present list of 1752, we are forced to the conclusion that astigmatism begins at the kindergarten age, say from six to ten, due to hypermetropia, at a time when near eye-work is very light.

Assuming that the primary cause of astigmatism is hypermetropia, we proceed to consider the unsettled question, "How is astigmatism produced?" The distinguished writers Fuchs, Juler, Donders, and Schmidt-Rimpler locate the seat of astigmatism in the cornea. Landolt says it is occasionally formed by the crystalline lens. Fuchs, Landolt, and others speak of astigmatism as congenital or acquired, either from irregular development or an irregular action of the eye. They all state that the greater curvature of the cornea lies closer to the vertical meridian than to the horizontal. When they say it is congenital, there is no more to be said; when they say it is acquired, some of them speak of the lids as the cause. They also speak of an increased curvature of the crystalline lens in the opposite axis to the greater curvature of the cornea, to correct the corneal astigmatism entirely in some cases and in others overcorrect it, as Landolt mentions and claims to have proved by the use of atropin.

That the crystalline lens becomes astigmatic in order to correct the corneal astigmatism is, we believe, universally accepted, but we are not aware that any one believes the crystalline lens produces it when there is no corneal astigmatism. The crystalline astigmatism is only a remedy for the corneal astigmatism.

The purpose of the lids in astigmatic cases is to cut off the end rays on the macula, so as to square the letters and make them more readable. The lids also trombone in myopia and high degrees of hypermetropia in order to cut off a part of the rays to diminish the

circles of diffusion. When these three classes of cases are corrected with glasses, the lids cease to trombone and furnish no evidence of having produced astigmatism. If the lids produce permanent astigmatism, this pressure would come directly above and below the cornea, thus making the vertical curvature greater. If this pressure were continuous, we might have some reason for believing that the lids cause astigmatism, partly closing to produce vision, if only for a moment.

The writer has endeavored to eliminate the three universally accepted causes of astigmatism in order to bring forward what he believes to be the true cause, namely, the external ocular muscles. The six are all opposing muscles, and all work together in accommodation. We begin life with hypermetropic eyes. A child's mental capacity at first is not strong enough to fix intently on any object, so no direct image is formed on the retina. When he gets away from his mother's lap and learns to walk, his mind is still not sufficiently developed to form a sharp focus on objects. This weakness may produce convergent squint, though not astigmatism, as the eyes are not fixed long enough on objects, and then they are constantly changing to distant objects.

When children reach the kindergarten age, as the hours are short and the study is light, only a few cases of astigmatism develop. Only 20 plus and 9 minus were found—a total of 29 of the 1752. During the school years from ten to twenty more concentration is required and longer hours, and astigmatism is increased to 82 plus and 58 minus, total, 140. From twenty to thirty, when a large percentage have entered the commercial world, and some are still in college, the work is harder, with longer hours. The number in this decade is 218 plus and 143 minus, total, 361. In the thirties the work is still more trying, as it is the time entirely of practical application of the knowledge they acquire in the second and third decades. The number is increased to 277 plus and 185 minus, total, 462. In the forties we find a slight decrease, to 230 plus and 146 minus, total, 376. This is accounted for by half of the fifth decade being in the presbyopic age. In this period we find the increased vertical curvature of the cornea decreased 11.6 per cent., while the obliques above axis 45 degrees and 135 degrees, including axis 90 degrees, decreased only 7 per cent. This means either that the axis is shifting from the vertical to the oblique above axis 45 degrees and 135 degrees, then later below and finally to the horizontal, or that the astigmatism disappears first in axis 90 degrees and second above the 45 degrees, and later on in life there is a decrease below axis 45 degrees until the astigmatism is almost entirely confined to axis 180 degrees for plus glass, and 90 degrees for minus glass.

If we are willing to admit that the four external ocular muscles supplied by the third nerve act in accommodation and the fourth and sixth nerves supplying superior oblique and external muscles counteract at the time of accommodation, then we are obliged to acknowledge that all these muscles act in correcting hypermetropia at the distance.

These muscles are inserted in the sclera near the cornea in such a way that any one muscle may have more or less a countereffect upon any other muscle, unless it should be the interni and inferior recti, which are from the same origin; still, in a way, they oppose each other as they pull downward and inward instead of directly downward or inward. The superior recti guide the inner and upper direction of the eyes to prevent them from being too much under control of the inferior recti. The inferior oblique acts in accommodation to counteract the superior recti and keeps the upper part of the eye in the vertical plane, just as it would be when at rest. The superior oblique relaxes to allow the inferior rectus to hold the eye below in its vertical plane, while the eye is turning downward and inward. In this particular action the superior and inferior obliques are allies, because the relaxation of the superior oblique would not

be sufficient if the inferior oblique did not prevent the eye from tipping inward above. On the other hand, the upward action of the inferior oblique can prevent too much torsion by the relaxation of the superior oblique. All these actions are to prevent torsion. Besides these, we have the direct opposing actions, the superior oblique against the superior recti, inferior oblique against the inferior recti, and externi against the interni, which is shown by paralysis of any single muscle, as the eye turns from that muscle in the direction of the opposite muscle.

We must keep in mind that hypermetropia is corrected from the time the eyes are opened in the morning until closed at night by accommodation for the distance, except in high degrees, when the eyes frequently blur to give the muscles a rest. Another exception is when the hypermetropia is so great that all attempts to correct it are abandoned. These cases become amblyopic. This hypermetropia runs from 5 D. to 7 D., and in some cases more, and vision is usually 20/100 or 20/70.

We have another class of amblyopia: compound hyperopic astigmatism about equally divided between the sphere and cylinder, total, 5, 6, or 7 D. These cases, in my opinion, struggle to correct the error, change half the error into astigmatism, without being able to accommodate to correct the remaining spheric part. This gives a double lined diffusion instead of circle diffusion. (The writer's explanation in his paper, published in 1894, on the changed spheric cornea to spherocylindric by the action of the muscles, will be the same here, as time has not changed his opinion.)

Our experience in taking the tension of an eye with the fingers shows us how easily the curvature of the eyeball is increased between two fingers. Why should not the curvature of the eyeball be increased by two opposing muscles? In fact, the insertion of these muscles is practically where the fingers are placed in taking the tension. If the pulling of the opposing muscles can have a like effect as the fingers, we not only have an increased curvature of the eyeball but of the cornea as well. As the eye approaches the presbyopic age the ciliary muscle has less effect on the eyeball, as it is gradually less soft and less yielding to the opposing muscles. Thus, the accommodation begins to subside and the astigmatic error changes into a spheric error. Many of the writer's private cases have demonstrated this fact.

The writer has collected an additional list of 1221 cases from his office records, which afford further proof that muscles cause astigmatism. In compound hypermetropic astigmatism, when the sphere is the same, we have corresponding axes in the astigmatism. The table shows this to be true in 90.4 per cent. against 9.6 per cent. different axes; also that the axes differ when the spheres differ. These cases show only 25 per cent. corresponding and 75 per cent. different axes. This proof holds good with both compound myopic and simple astigmatism; also in mixed, when spherocylindric glasses are ordered the same or different.

We sometimes overcorrect the astigmatism at the expense of the spheric or the reverse, as the eye may see the same with a slight overcorrection of the other, but to increase one without diminishing the other reduces the vision. If this be true, the theory advanced may be wholly true. The reason as it looks to the writer is this: The same sphere requires the same accommodation which causes a corresponding axis. Anisometropia, requiring more accommodation in one eye than in the other, causes a torsion of that eye which makes a different axis; or if the accommodation is suspended in that eye, a torsion with axis different ensues on the reverse side.

In other papers the writer has stated that when astigmatism exists, whether the same, different, or only in one eye, the hypermetropia, if any, will be found to be the same, with few exceptions. The usual exceptions occur when there is a decided difference from 1 D. to 4 or 5 D. This is true also with no astigmatism. Only 3 cases were found in 19 that had less than 1 D.

difference in simple hypermetropia. With astigmatism only 5 in 17 cases with less than 1 D. spheric difference. Total of 36 cases, 28 more and 8 less than 1 D. difference, 78 per cent. and 22 per cent. The apparent reason for this is that if astigmatism develops in anisometropia, it begins first in the eye of the greater hypermetropia by changing the difference into astigmatism, making the spheric part the same. If astigmatism is brought about by excessive accommodation, it comes first in the eye with the greater error, as it requires more effort to correct this eye by trying to equalize the vision of the two eyes. This fails when the difference is too great. That is why we seldom see the difference less than 1 D. If any further astigmatism develops, it is the same in both eyes, leaving the spheric error still the same.

Among 23 con. squint cases, all those found under the age of six were spheric only. The only astigmatic cases were ages ten and eleven. These cases were straightened by prescribing the ophthalmoscopic measurement, and verified with the test-lenses in only the two astigmatic cases. Only in one was atropin used, and that was in the astigmatic case aged eleven. The only other case above the age of the two astigmatic cases was aged sixteen, which may have been tested with the lenses; but, as it was not an astigmatic case, +4 D. was ordered, as found by the ophthalmoscope. The other 20 ages ranged from nine years down to twenty-two months.

In looking over the anisometropic eyes and same spheric error to see what influence each had on the axis of the astigmatism, the same books were examined, and the cases with no astigmatism were copied to get the percentage of same sphere and different. The result follows:

Spherical, same, 1,433 cases.....97½%
Spherical, different, 38 cases..... 2½%
Spherocylinder, same, 372 cases.....94 %
Spherocylinder, different, 26 cases.... 6 %

In conclusion the writer restates what he has attempted to prove by the several groups of cases in the present findings of 2,974 astigmatic and 1,818 spheric cases—4,792.

First: Astigmatism is not congenital, not acquired by the crystalline lens, and not by the action of the lids; because none of them would start the greater curvature of the cornea in the vertical axis at 100 per cent. under age ten and gradually change the axis of the greater curvature 100 per cent. in the horizontal axis at the age of seventy. This evolution is complete at the end of the ciliary muscle action. After this age, when the ciliary muscle has ceased to accommodate, the external muscle cannot assist in accommodation, but acts only in convergence and in associated movements.

Second: The axis of astigmatism is largely controlled by the sphere; that is, the axis corresponds when the sphere is the same and differs when the sphere is different. In the latter case we may find torsion, but not when the sphere is the same. The exception to this rule is small.

CAUSE OF MYOPIA.

It is an accepted fact that hypermetropic eyes become round or nearly round at full growth, and in some cases myopic. In myopia the increase in the anteroposterior diameter is thought to be due to the thinning of the sclera posteriorly. This has been demonstrated by microscopic examination on sections of the sclera, as well as by our ophthalmoscopic findings. The writer believes that this posterior thinning begins in the advanced stages of myopia, due to a tendency to diverge, with a more rapid increase in actual divergence. In low degrees of myopia the fundus usually looks normal, and we seldom see posterior staphyloma in eyes of less than 8 D. The sclera when normal is thicker posteriorly and gradually becomes thinner as it approaches the cornea, so the thinnest part is anterior to the insertion of the six muscles. It is quite probable that the constant correcting of hypermetropia in children is not entirely by the ciliary muscles, but is aided by the six external ocular muscles attached to the sclera at a corresponding distance from

the cornea. It seems reasonable to suppose that these muscles just back of the thinnest part of the sclera would depress that part of the sclera including the cornea, producing in a measure conical cornea; that is, a deeper anterior chamber. Hypermetropic eyes that develop into emmetropic at maturity may be due to this muscle action, or at least influenced by it. Children who are obliged to hold their book at 8 inches or less to see the print increase the accommodation 5 D. on probably 4 or 5 D. distant error. This great strain to correct 10 D. may start a low grade of sclerochoroiditis which may be the starting-point of myopia. Those of hereditary tendency are more easily influenced. This thinning of the sclera is occasionally indicated by a choroidal ring about equal distance from the insertion of the muscles and the cornea.

HYPERMETROPIC ASTIGMATISM—EVOLUTION IN THE GREATER CURVATURE OF THE CORNEA FROM THE VERTICAL TO THE HORIZONTAL AXIS.

Age	Total	Ax. V-VO	Ax. V	Ax. VO	Ax. 45 135	Ax. H-HO	Ax. H	Ax. HO	Per cent V-VO	Per cent V	Per cent OV	Per cent 45 135	Per cent H-HO	Per cent H	Per cent H
6-10	20	20	10	10	0	0	0	0	100	50	50	0	0	0	0
10-20	82	68	38	30	0	14	14	0	83—	46.34	36.58+	0	17	17	0
20-30	218	156	77	79	11	51	29	22	71.5	35.3+	36.2+	5	23.5	13	10.5
30-40	277	214	120	94	13	50	26	24	77	43.32	33.92	4.6	18	9.3+	8.6+
40-50	229	161	73	88	8	60	47	13	70	32.3	38.4	3.4+	26	20.5+	5.6+
50-60	168	75	43	32	7	86	55	31	44.6	25.5+	19+	4+	57.1	30	18.4+
60-70	67	16	5	11	4	47	31	16	23.8	7.4+	16.4+	5.9+	70	46.2	23.8+
70 up	38	4	0	4	0	34	30	4	10.5	0	10.5	0	89.4	78.9	10.5
	1099			8											

MYOPIC ASTIGMATISM—EVOLUTION IN THE GREATER CURVATURE OF THE CORNEA FROM THE VERTICAL TO THE HORIZONTAL AXIS.

Age	Total	Ax. V	Ax. OV	Ax. V-OV	Ax. 45	Ax. OH	Ax. H	Ax. H-O	Per cent V	Per cent OV	Per cent V-OV	Per cent 45	Per cent OH	Per cent H	Per cent H-OH
6-10	9	.6	3	9	0	0	0	0	66⅔	33⅓	100	0	0	0	0
10-20	58	17	30	47	1	8	8	10	29.3	51.7+	81	1.6	13+	3+	16.4
20-30	143	34	46	80	6	27	30	57	23.77	32.17	55.94	4	20—	20+	40—
30-40	185	67	48	115	11	23	36	59	36++	26+	62+	5.7	12.4+	19.4	31.8
40-50	156	38	46	84	11	24	27	51	24.3	29.5	53.8+	7	15.3+	17.3+	36.2+
50-60	65	19	14	33	4	9	19	28	29.2+	21.5+	50+	6	13.8	29.2+	44
60-70	23	5	3	8	0	4	11	15	21.7	13	34.7	0	17.3	47.8	65.2
70 up	13	2	2	4	0	4	5	9	15.3	15.3	30.6	0	30.6	38.4	69
	652														

This list will help in confirming the first list that astigmatism is caused by the external ocular muscles, by the spherical error influencing the axis of the astigmatism.

COMPOUND HYPEROPIC ASTIGMATISM.

	No.	Per Cent.	Total
Same sphere, corresponding axis	336	90.4	372
Same sphere, different axis	36	9.6	
Different sphere, different axis	18	75.0	24
Different sphere, corresponding axis	6	25.0	

HYPEROPIC ASTIGMATISM.

Hyper. same cylinder. Ast. corresponding axis	203	90.6	244
Hyper. same cylinder. Ast. different axis	21	9.4	
Hyper. different cylinder. Ast. different axis	38	39.2	97
Hyper. different cylinder. Ast. corresponding axis	59	60.8	
Total carried forward			737

COMPOUND MYOPIC ASTIGMATISM.			
Same sphere, corresponding axis	70	73	96
Same sphere, different axis	26	27	
Different sphere, different axis	76	60.8	125
Different sphere, corresponding axis	49	39.2	
MYOPIC ASTIGMATISM.			
Same cylinder, corresponding axis	73	92.4	79
Same cylinder, different axis	6	7.6	
Different cylinder, different axis	14	20.2	69
Different cylinder, same axis	55	79.8	
MIXED ASTIGMATISM.			
Converted to same + sphere, corresponding axis	31	77.5	40
Converted to same + sphere, different axis	9	22.5	
Converted to same — sphere, corresponding axis	21	65.9	32
Converted to same — sphere, different axis	11	34.1	
Converted to + and — spheres, corresponding axis	7	46.7	15
Converted to + and — spheres, different axis	8	53.3	
Sphere not same, corresponding axis	13	46.5	28
Sphere not same, different axis	15	53.5	
			1221

CONSERVATIVE TREATMENT OF ASTHENOPIA IN CHILDREN.*

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During a five years' study of refractive conditions among our school children, the need of conservative treatment seemed to me most important. Unfortunately, it is a subject which has been somewhat neglected and considered decidedly uninteresting. Both public and private school pupils were treated. A more or less successful attempt was made to keep in touch with these pupils by yearly examinations, watching constantly for any new complications. Office methods were copied in the hospital clinic, as closely as conditions allowed.

Considerable extra time and pains were taken to give hospital patients as good advice and treatment as possible. Palliative treatment cannot be carried out quickly; it is not possible to dispose of all cases in one or two visits. It requires, first of all, a careful assembling of data in order that an exact diagnosis can be made. To understand the complaint of the child it is quite essential to consult with the parent or the school nurse. Only in this way can we decide, in doubtful cases, whether glasses are necessary or whether some other better alternative treatment will bring the desired result.

The children were divided into three groups—

Group 1. Pupils usually brought by the school nurse. who had high refractive errors requiring immediate correction with glasses.

Group 2. Scholars having a small—less than one-half diopter—refractive error, with defective vision of one eye; who sometimes complain of indefinite symptoms, and who very often require glasses as well as general medical treatment.

Group 3. Children brought to the clinic or office by the parent very often present more difficult problems. Frequently such children are brought for an eye examination not because their vision was found defective, but on account of some slight eye symptoms which suggested eye-strain.

Group 1. These children need glasses prescribed immediately in order to preserve vision from getting any worse; also to prevent strabismus. Only by this means can they progress steadily with their education.

*Read before the American Ophthalmological Society, July 10th, 1918.

Group 2. About 75% of all the scholars of this kind require glasses to relieve very definite symptoms of asthenopia. A majority should receive a combined treatment to relieve the eyes and other obvious defects; as adenoids, diseased tonsils, decayed teeth, etc. About 20% of this group never received glasses.

Group 3. This includes children generally brought to the clinic by the mother. They may or may not have been notified that the patient had failed to pass the school test. Very often the pupil comes because the parent has observed that headaches start after studying.

Conservative or palliative treatment can be only properly undertaken when careful tests show the following conditions exist:

A. A very small refractive error carefully determined under a mydriatic. A comparative test was always made without drops. In my office two independent estimations of the refraction were made, but in the clinic the retinoscopy was generally not attempted without drops, as it takes too long, and our inexperienced house-officers have rarely had an opportunity to try this procedure.

B. The original test of vision, let us say, disclosed a decrease of one eye only. It was reduced to 20/40 at least, in order to attract the teacher's attention. The school tests usually were found correct. My own tests tended towards a slightly higher average because they were more carefully done under a little better conditions.

C. Examinations by the medical or throat consultants at the hospital showed other physical ailments, such as malnutrition, indigestion with constipation, bad teeth, diseased tonsils, and adenoids. Concerning these complications among private cases, it was necessary to get the data from the family physician.

Conservative treatment is justified if we admit that a temporary lowering of vision may accompany the different milder types of chronic conjunctivitis or even hyperemia of the conjunctiva. If persistent conjunctivitis is cured with drops, the vision improves and the child may then be able to pass the required visual examination.

This axiom has been one of my guides in the treatment of many children. It is so simple, and at the same time so effective, that often the child requires no further treatment. It seems to me that it should be tried in a great many more cases, but I know that few of my colleagues will agree. The reverse principle has guided most ophthalmologists that, having brought corrected vision to normal with proper glasses, all other ocular complaints would cease. A few of our members will agree that special emphasis should be laid on a principle of conservative treatment, which is directly antagonistic to the views of the majority. It is based on the broad, common-sense idea that glasses for many an average child, of the special group we are considering, should not be the one and only treatment. General practitioners outside of certain larger cities have been guided by this principle. The possibility that these doctors might do harm to the pupils' eyes by ignoring dangerous asthenopic symptoms has been eliminated by the ease with which oculists of nearby smaller towns can be consulted.

Special care was taken to separate the backward scholars in order to give them particular advice. If a child is in its proper grade at school and complains of indefinite asthenopic symptoms, conservative treatment is indicated. A much more thoro search for the underlying cause of complaint must be made, however, if the child is two or more grades behind the average. It makes no great difference if the scholar is one grade behind the average. A large proportion of public school children finish their education upon graduating from the grammar school. Several reasons, aside from physical defects, may explain why they are apparently backward, the most important of which are:

A late start, when almost 7 years old.

A change of residence with a consequent change of school and a readjustment into a different class.

Sickness in previous years, but which had left no bad sequelae to affect the scholar's subsequent progress.

On this account, I divided all the children into two groups:

Group A. Pupils in proper grade or one grade behind. (84%.)

Group B. Pupils two or more grades behind. (14%.)

If a scholar is in Group B, it is the duty of the physician to work with the educators to eliminate as rapidly as possible all physical defects. By so doing a determined effort is made to promote the child to a higher class or at least keep it from falling any further behind. The experience of one of our school nurses is that if a scholar once falls behind, an advancement into a higher class is next to impossible. The best that she expected was to report that the child was holding its own without further complaint, if all medical treatment was carried out faithfully.

Our aim is to improve both the vision and the general ability of the scholar to continue its studies by removing any evident physical defect. Palliative treatment means, of course, that a larger proportion do not receive glasses than in either of the other two groups. This method is used working on the assumption that so-called eyestrain can sometimes be treated by a combination of general medical treatment and the cure of conjunctival irritation by suitable collyria.

A moderate degree of chronic conjunctivitis often proves the most important cause of asthenopia. Also a conjunctival irritation, by causing the pupil to think about his eyes, will sometimes disclose a chain of symptoms which have no apparent relation to the eyes. For example, the child complains of the eye aching. This is often described as a headache. This does not always prove to be a characteristic frontal headache, because small children very often cannot describe accurately this symptom without some further questioning by the doctor. Hemicrania or pain in the eyes seems often the same thing to the patient. To differentiate between the two complaints we must wait until a careful refractive examination has been made before we decide if headache is likely to result from a very small refractive or muscular error.

Let us suppose the pupil complains of a frontal headache with intermittent attacks of pain and tenderness of the eye-balls. This starts two or three hours after the noon meal, at about the time school closes. The headache seems to come after studying. A careful refractive examination is made without coming to any definite conclusion. The diet is investigated and we discover that constipation and intestinal toxemia have caused the headaches. The medical consultant treats these conditions and the general health of the patient promptly improves. In the meantime, soothing collyria having been prescribed, we find often a perceptible improvement of vision. If the first visual test proves the child unable to meet the State requirement, the above treatment will remedy the defect. Mental dullness changes to alertness after freeing the intestines of a number of poisons. At the same time, by relieving conjunctivitis, the child's attention is withdrawn from his eyes and it proceeds with all studies in a normal manner without further complaint.

If we have found only insignificant physical or ocular defects present, we can now consider a very interesting mental attitude of some scholars towards apparent asthenopia. We realize how easily suggestion will affect a cure of the childish mind. A resignation to the inevitable will also stop all complaints. This is shown very well by observations made by one of the teachers at a large boarding-school for girls in this state. A certain number of the pupils complained of headache and other asthenopic symptoms and were sent me for examination. For some of the girls glasses were prescribed, and they started again with their studies.

It was a rule that only the senior class could study in their rooms, an arrangement much envied by

their younger schoolmates. The lower classes all gathered together to study, under the supervision of a teacher, in a large hall. The girls claimed the illumination of this hall was insufficient and that it was a frequent cause of the asthenopia. Most of the younger scholars, for whom glasses had been prescribed, soon became resigned to working under the above conditions and ceased fretting. Also it was noticed that they very often discarded their glasses and made no further complaints. We must acknowledge that any child is liable to act in just this manner.

Unless strict supervision by their elders is observed, the child may take off his glasses, especially in cases where hyperopic errors have caused the headache. The vision without glasses being very good, although possibly not perfect, and the headache stopped, the scholar will often experiment to see if he cannot get along without them. The oculist is deluded by thinking that his patient is carrying out his instructions. The ability of the pupil to reestablish his equilibrium, so to speak, has been ignored. In these cases glasses should not have been prescribed except as a last resort in case all so-called temporary asthenopia could not be relieved by using collyria, or, more often, some general medical treatment.

The need of giving a plus glass to a child to correct temporary accommodation weakness seldom arises. A series of experiments made upon both youths and adults convalescing from pneumonia and typhoid — these diseases chosen on account of the great drain on the patient's vitality—showed a wonderfully quick recovery of the accommodation. Tested one or two days after the crisis of the fever had been passed, the accommodation was generally reduced to one-half of normal. This reduction was in direct ratio to the patient's youth. Within two weeks the accommodation was again normal. Debility following such serious diseases, however, makes the child complain of eye-strain even from very small refractive errors.

The need of giving temporary relief with glasses should not be overlooked. This method of treatment is, of course, very generally employed. What is overlooked, however, is a decided tendency of a child to put aside the glasses as soon as symptoms cease, or anyway as quickly as experiment shows that no symptoms recur if the glasses are discarded during study hours. As mentioned before, this childish act is inevitable, and certainly very often it does no harm. During convalescence from some of the children's diseases—especially measles—the skillful prescribing of suitable collyria will suffice. At such times the child's complaints are apt to be more specific, and we must not fail to give every help to tide the scholar over a difficult period. The length of time that help with glasses or other treatment is needed varies in direct proportion to the child's recuperative power. A neurotic temperament is a complication which prolongs the treatment.

A few scholars are brought by the nurse or mother on account of apparent poor vision of one or both eyes. The failure of the pupil to pass the school tests may be due to shyness or nervousness, or to improper illumination of the test letters. Sometimes my tests did not confirm the previous ones. With such scholars it is often extremely important for the parent to report the actions of the child at home. Attention should be directed to any of the following characteristics:

a. Lack of concentration and good application;

b. Missing entire words or mispronouncing words when reading aloud;

c. A tendency to hold the book too close—simulating myopia.

d. Bad sitting posture;

e. Eyes or lids inflamed;

f. Too rapid appearance of fatigue, causing listlessness or inattention.

A temporary decrease of accommodation may follow very vigorous exercises, like football or rowing. Experiments to demonstrate such a condition have been recently begun, and in the future I shall hope to have more information to report.

A small decrease of vision always accompanies the lessened accommodation. This condition is presumably responsible for a good deal of the lack of concentration and inability to do good work during study hours at home in preparing home lessons. Such an artificially produced accommodation fatigue of a child may be compared to the easily demonstrated tiring of adult eyes after a busy day. These tests are especially hard to perform, because the child soon lacks interest and then does not make the maximum effort required. The best method seems to be to take a very small number of children and train them sufficiently to observe slight changes of visual acuity. If a series of tests made in the evening show a decrease, control examinations the following morning are necessary. My own observations have proved that my accuracy of observation was influenced often by mental conditions. If the mind is dulled by loss of sleep, nervous fatigue, etc., the test becomes proportionately more difficult. Brain fatigue more often caused a decrease of accommodation than physical exercise.

SOME OBSERVATIONS ON ASTIGMATISM.

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Remarks on the terminology of astigmatism especially direct and inverse, and on the frequency of the latter, with discussion of practical points in dealing with astigmatism. With three charts. Read before the American Academy of Ophthalmology and Oto-Laryngology, August 6th, 1918.

Astigmatism has generally been divided into direct, or astigmatism with the rule (meridian of greatest curvature vertical or nearly so), inverse, or astigmatism against the rule (meridian of greatest curvature horizontal or nearly so), and oblique astigmatism (meridian of greatest curvature in the vicinity of 45° or 135°). Some writers omit the third subdivision and classify these oblique astigmatisms with the first group. As we[1] have recently pointed out, this classification is purely arbitrary, as these cases might with equal reason be classified with astigmatism against the rule.

It is singular that an exact terminology has not been adopted for this most exact subdivision of ophthalmic science. One has only to peruse the chapters on refraction in our text books to learn that each writer has felt at liberty to make his own definitions, which are generally characterized by a vagueness incompatible with scientific precision. We note that even Jackson[2], whose careful study of changes in astigmatism is so illuminating, speaks of direct and inverse astigmatism with meridians of greatest curvature "approximately" vertical and horizontal.

In order to eliminate any uncertainty in the future as to what are the boundary lines of the three groups, we propose the following definitions:

Astigmatism with the rule, or direct astigmatism, shall include all cases with meridian of greatest curvature at any point on the arc from 46° to 134°.

Astigmatism against the rule, or inverse astigmatism, shall include all cases with meridian of greatest curvature at any point on the arc from 0 to 44° and from 136° to 180°.

Oblique astigmatism shall include all cases of astigmatism with meridian of greatest curvature exactly at 135° and 45°.

It has been argued that there is no necessity for any subdivision, for the reason that practically every eye possessing an astigmatic error should wear a cylindrical correction and that it is immaterial whether the meridian of greatest curvature is in the vicinity of 90° or 180°. Theoretically, this contention has merit. But we are convinced that it is useful, on clinical

grounds alone, to differentiate at least between direct and inverse astigmatism, for the reason that the latter presents certain features of special interest and importance.

Our study indicates that inverse astigmatism is more frequent than is generally surmised (in 1024 eyes with regular astigmatism we found 283, or 27.7%, inverse). Furthermore, as Theobald[3] has pointed out, an eye with this type of error is much more capable of "hiding it away" than is an eye with direct astigmatism. This observation accords with our own experience. We are convinced that prior to our systematic use of Lancaster's astigmatic charts many examples of low inverse astigmatism eluded us.

In our series, too, we determined by neutralization of spectacles prescribed by ophthalmologists who had preceded us that more than half of the men had failed to incorporate a cylinder to correct an inverse astigmatism. Glasses prescribed by refracting opticians only very rarely indicated any appreciation of the existence of this type of error. Low inverse astigmatism gives rise to especially annoying symptoms, such as headaches, blepharitis, congestion of the globe, etc. It is less well borne by the eye than an equal degree of perpendicular astigmatism.

One of the principal reasons for the greater degree of asthenopia in eyes with inverse astigmatism is the greater interference with the legibility of ordinary printed type. In direct astigmatism the vertical components of letters are distinct, whereas the opposite is the case in inverse astigmatism. Letters with vertical components distinct are more easily recognized than those with horizontal components distinct. One has only to render oneself artificially astigmatic with the rule and then artificially astigmatic against the rule (using the same cylinder) to appreciate the difference in distinctness of the same line of letters. This difference can also be beautifully demonstrated by the camera. In the illustration, the upper group of letters was photographed with an ordinary lens, the second group with a lens rendered astigmatic with the rule, and the third group with a lens rendered astigmatic against the rule (using the same cylinder). No one will fail to appreciate the lessened legibility of the third group. See Fig. 1.

It is probably an accurate statement of fact that one finds frequently that for which one is on the lookout. This is exemplified in the search for astigmatism, and particularly for inverse astigmatism. That we all possess some amount of astigmatism is a truism; whether it be corneal or lenticular is beside the point. Having detected a small amount of astigmatism against the rule, it does not follow that it should be corrected in all instances. In the treatment of this condition the exercise of judgment is as necessary as in all refractive work.

In the correction of refractive errors there are two things to be subserved, viz., improvement of vision and the increased comfort of the patient. If clarity of vision be gained without comfort, or comfort obtained with no improvement in vision, the result has fallen short of the ideal. The average patient will greatly prefer a correction which yields fairly good vision with perfect comfort to that which gives the most acute vision with an aggravation of his discomfort. And occasionally this is just the effect of incorporating a cylinder in the correction. We feel, therefore, that for some patients, especially elderly ones, who for years have been completely comfortable with spheres, it is ill advised to prescribe an astigmatic correction which, theoretically, is indicated. The confusion and discomfort incident to the new order of things offset any increase in visual acuity. Many of us are creatures of habits, and even of bad habits.

On the other hand, when it is definitely demonstrated that both vision and comfort are increased by an astigmatic correction, it is incumbent upon us to determine with all possible accuracy the strength and axis of the cylinder. And this may necessitate the use of cycloplegics in certain individuals above the age when such drugs are usually used. We believe this to be

good practice. We do not believe that a drug like homatropin ever produces glaucoma. It may precipitate an attack, but does not cause the disease. It goes without saying that a careful exder cycloplegia when the precycloplegic test has given unsatisfactory or conflicting results. It might appear superfluous to labor this point were it not for the fact that a large number of

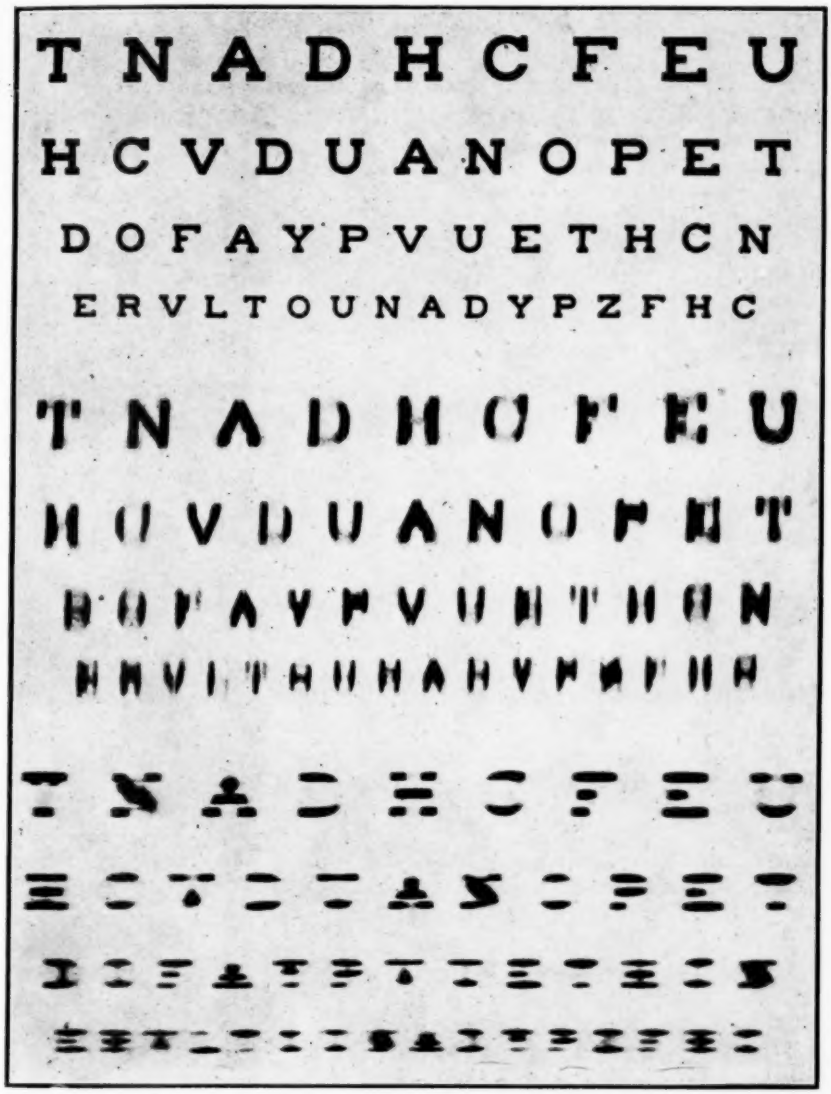

Fig. 1. Test letters photographed: The upper set with spherical lens giving images like those of the normal eye. The second set through cylindric lens imitating direct astigmatism. The bottom set thru cylindric lens imitating inverse astigmatism.

amination of the fundus, tension and field should precede the use of the cycloplegic in these cases. Hence the presbyopic patient need not be deprived of the advantages of a test unprominent oculists are unalterably opposed to the use of cycloplegics in adults of whatever age.

The advice of Clarke[4], that the full amount of astigmatism should be cor-

rected, cannot be followed in all cases with comfort to the patient. In numerous instances the postcyclopegic test reveals the fact that the patient is either made uncomfortable with the full cylinder or that the visual acuity is reduced. In either event a glass of less cylindric strength must be given. For instance, a plus 1.5 cyl. of a total of 2.5 may be all a patient will accept at the first test. Some time later plus 2 is accepted, and finally the full cylindrical correction is worn with comfort. It is not to be inferred that the astigmatism in such cases has changed. These facts have recently been emphasized by Col. Elliot[5].

One must be careful not to overcorrect an astigmatic error even in the slightest degree. It is better to err in the opposite direction. Wipper[6] has recently stated that a patient should be left with a quarter diopter of astigmatism with the rule uncorrected, as this is the normal condition and even essential for comfortable, if not perfect, vision.

Always a difficult problem is the management of cases of mixed astigmatism in which there is accommodative cramp. It is very disconcerting to prescribe a glass on the basis of a cycloplegic measurement, only to find that the patient complains bitterly of poor distant vision. Various measures, such as prolonged atropinization, rest of the eyes, change of occupation, sedatives, etc., often do not suffice to overcome the exalted tone of the ciliary muscle. It has seemed wise in some cases of this kind to yield to the demands of the patient for good distance acuity, by increasing the strength of the minus spherical element. We have not had cause to regret this departure from scientific accuracy.

Not rarely do we encounter cases in which one eye shows a low regular astigmatism, whereas the other eye presents a high degree. The cause for this discrepancy is often obscure. Without doubt a number of unilateral cases of high astigmatism are the result of trauma occurring at birth.

The following is an illustrative case:

In 1912 Baby, L. O., was seen by one of us four days after birth. The obstetrician stated that one blade of the forceps had pressed firmly over the child's left eye. The cornea was entirely opaque, but there were no inflammatory signs. Under dionin and hot applications, the cornea cleared.

A few days ago, this little girl, now six years old, was brought to us because the mother noticed that the child in looking at picture books had a tendency to close the left eye. Right vision 6/5. Left vision 6/40. The ophthalmometer showed: R. As. 1 mc, 90; L. As. 6 mc, 90. Both corneas were perfectly regular.

Oblique illumination showed the presence of three vertical striae situated in the deeper layers of the cornea, one coinciding with the inner pupillary margin, the second with the outer pupillary margin, the third midway between the latter and the outer limbus. A skiascopic examination disclosed a regular astigmatism plus 4.5 D with meridian of greatest curvature at 90°. With plus 4, cyl. axis 90°. V. 6/10.

Every one possesses a small amount of irregular astigmatism which is, of course, not perfectly correctable by any cylindrical lens. Whenever there is a deformation of the cornea from injury or inflammation, or when the lamellae of the lens are irregularly disposed or swollen, as in incipient cataract, there is bound to be more or less irregular astigmatism which may or may not be associated with the regular type.

In the presence of irregular astigmatism, whether of corneal or lenticular origin, it is thoroly worth while to determine with the greatest care the cylindrical or sphero-cylindrical combination which yields the highest vision. When the irregular astigmatism is a more or less fixed quantity, as, for example, in an eye with a cornea deformed by a long past keratitis or injury, a painstaking correction with sphero-cylinders will often be highly acceptable to the patient, who is grateful for the heightened acuity thus

secured. But every case is a law unto itself; not rarely a simple sphere, which yields less vision, is preferred to any combination.

An eye with an incipient cataract which has previously rejected all cylinders, may often be made to see very well with a sphero-cylindrical combination. But here again it is often a case of "Love's labor lost"—the combination is soon found uncomfortable or visually unsatisfactory. We presume that the explanation lies in the fact that the irregular lenticular astigmatism is constantly changing with the progress of opacification.

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RECONSTRUCTION AND RE-EDUCATION OF DISABLED SOLDIERS AND SAILORS.

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Number of different disabling injuries and what is being done for them in this and other countries. Read before the American Academy of Ophthalmology and Oto-Laryngology, August 5th, 1918.

We all know what happened to the soldier and sailor disabled in our former wars. After a few brief weeks of hero worship, marked, perhaps, by a kindly tolerance of his oft-told tales of adventure in the army and on the battlefield, he was given a pension, and in some old soldiers' home or as a dependent upon the semi-charity of neighbors or relatives, eked out a half-forgotten existence, a bore to himself and useless to his country. It is true that a few of these returned invalids, in spite of their handicaps, resumed their former occupations or adopted some new industrial activity, but their number was so small as to be almost negligible.

As a matter of fact, the obstacles that lie in the way of a resumption of industry after the combatant has passed weary months in the field and in various hospitals, are to the sick or wounded man almost insurmountable. Unless prompt and continuous help is given, he lapses into a state of discouragement from which it is difficult to arouse him.

It has long been recognized that to be most effectual this mental and moral assistance, this "cheer up," this achievement of victory over wounds should begin at the earliest possible moment. It should be prearranged and definitely organized. All the belligerent countries have attacked the problem from this viewpoint and by conducting, chiefly through army nurses and medical officers, a campaign of education, early teaching the disabled man that his condition is not as hopeless as he probably thinks it is; and that the Surgeon General and other governmental agencies will see to it that his disablement will be reduced to the minimum, that he will be refitted for industry and that his family and he will be supported while he is undergoing reconstruction. He will be further assured that his war risk insurance compensation for disability will not be reduced as a result of his increased powers through reconstruction. Finally, a desirable job will be found that will enable him to become an independent, wage-earning

citizen, taking part in and enjoying the daily life of his own community.

This verbal campaign is further strengthened by posters displayed in the hospitals and on board ships returning from overseas, by cards entitled, "What every disabled soldier and sailor should know," by booklets telling how, when and where physical and vocational restoration is accomplished, and last, but not least, by moving pictures such as I expect to exhibit to you this evening.

In these and other ways we spread the good news that there are really no longer any "cripples," but only a readjustment of the soldier's reduced powers to altered conditions; and that the so-called disabled man can, if he wishes, improve and apply these powers, that he may even adopt a trade or profession more congenial or more lucrative—or both—than he followed before his injury or illness.

Who, let us now ask, are the war-disabled requiring rehabilitation?

Naturally one thinks of those who have lost eyesight or hearing or a limb, and these cases are of great importance and require special attention, but they really make up only about one-third of the whole number of the handicapped. We must not forget the tuberculous, constituting a large percentage of the total; the nervous patients, including the so-called "shell-shock" cases; the insane (curable and incurable); those with serious diseases of the heart; those that have been "gassed;" those with rheumatism, and many other internal affections. Any of these may suffer from a handicap even worse than the loss of a leg or an arm.

An approximate idea of the causes of disablement in the service may be obtained from a table based on statistics of the British army and navy during the present war. The figures indicate the number of cases in every thousand of disabled men, excluding officers:

Injury to eyes	32
Amputation of leg	30
Amputation of arm	19
Injury to leg not requiring amputation	121
Injury to arm not requiring amputation	82
Injury to hand not requiring amputation of whole hand	61
Injury to head (including six or seven cases of paralysis)	47
Hernia	7
Miscellaneous wounds and injuries (including about five cases of paralysis)	53
Total wounds and injuries	453
Disease of chest (including, say, 60 cases of tuberculosis)	124
Rheumatism	50
Diseases of heart	110
Epilepsy	11
Nervous cases	47
Insanity	9
Deafness (partial, or in comparatively rare cases, total)	26
Frostbite	10
Miscellaneous diseases (among which may be noted, besides a few cases of paralysis, Bright's disease, ulcer of stomach, debility, and varicose veins	160
Total causes of disease	547
Total wounds and injuries	453
	1,000

It may be added here that the number of men requiring extended treatment and vocational training constitute a relatively small proportion of the total number of those discharged from the army or navy. Although soldiers and sailors suffering from certain chronic diseases, such as tuberculosis, insanity and other mental and nervous disorders, may require treatment for a considerable period under special conditions, yet the time of retention in army hospitals after return to the United States will be relatively short. Some will be so nearly well as to permit of their discharge almost immediately upon landing. A considerably larger number will require treatment for a week or even a month or two in the general hospitals. About 90 per cent of the returned disabled men will be discharged after a comparatively brief period of treatment, and will require no special measures of physical or vocational reconstruction.

Our Government has already provided ample hospital accommodations for all these injuries and diseases, and hundreds of soldiers are now being restored to health through the agency of the Surgeon General and his medical officers, and receiving, meantime, physiotherapeutic, including occupational and other forms of prevocational training.

When the soldier or sailor has been as far as possible restored to health and is incapable of further military service, he is discharged from the army or navy and

transferred to the governmental civilian agencies, especially the Federal Bureau for Vocational Education appointed by Congress for the completion of his education or re-education.

It is estimated that the proportion of the total number of disablements calling for re-education for an occupation different from the one formerly followed by the disabled combatant is about 10 per cent. In other words, under the care of the Surgeon General will fall about 90 per cent of returned invalids requiring reconstruction, including those who will also under him complete their occupational career. When discharged from the army cured, as nearly as may be, of their injuries or disease, most of these are expected to pass directly into industry, following their old or a new occupation.

The task of finding a suitable job for returned invalided soldiers and sailors, and of exercising supervision over them after their return to work, will devolve on the Federal Board of Vocational Education, on the War Risk Bureau, and probably, also, on the American Red Cross.

As has been pointed out by Mr. Lakeman of the American Red Cross and others, "the reconstruction of the disabled soldier is, in the first place, a matter of public duty which is called for by every consideration of national gratitude and justice. This alone is a sufficient reason for the fullest expenditure of needed effort and funds. But also sound national economic policy requires that the state, in order to conserve industrial and labor resources, should make skilled workmen of its disabled soldiers and find them employment. After this war every soldier who returns to a condition of idle dependence on Government support will help to diminish the productive power of the nation. With the terrible wastage of men and materials, every ounce of constructive energy will be needed to repair the damage and to face the renewed intensity of competition in trade and industry. In this country, the channels of immigration are closed during the war, and the draft on the labor market is unprecedented. The supply of skilled labor is no longer inexhaustible. In the field of industry, as in that of natural resources, the era of waste and extravagance is over and every element of productive man power must be conserved as an indispensable national asset."

There is, however, another argument in favor of recalling the disabled man to a useful life. If the handicapped soldier or sailor, for any reason, fails to make use of his industrial calling—fails to take advantage of his opportunities—he becomes a part of the nation's human scrap heap, an unhappy victim of his own and perhaps of his family's indolence and neglect. It is the salvation of a soul as well as a body for which the rehabilitation activities are striving, and for the accomplishment of which we ask your assistance.

The disabled soldier's decision as to his future must be backed up by the unhesitating support of his community. No system of governmental or private vocational education can succeed unless the man's will to succeed is stimulated and his ambition aroused by a well developed public opinion. No Government bureau can exercise authority over the man's personal will to make or neglect his future. For example, it is easy to imagine what a powerful influence the supporting opinion of the family and of the neighbors may be in inducing a man to forego the false glitter of an immediate job in favor of the sound metal of thoro training for an independent future. To convert the man to the wiser view, it will sometimes be necessary to convert his family first. Certainly his friends, relatives and companions must believe in the desirability of this victory over wounds, and this will to conquer difficulties, if the disabled man is to be successful in the contest with the powers of darkness.

It is not possible in one evening to enter into details of the therapeutic care and training now being given to even the most important disabilities of our returned invalids, but I would like to say something more about the war deaf and the blind.

The blinded warrior makes a strong appeal to the emotions, although perhaps for this very reason the extent and importance of this form of disability has

been exaggerated in the public mind. The French experience indicates that less than one-half of 1 per cent of the soldiers wounded in battle are blinded, the reason being, probably, that injuries sufficient to make a man blind in both eyes generally prove fatal.

Some of the foremost educators of the blind have co-operated with our Government in devising, perhaps, the most complete system of re-education and after-care of blinded soldiers which has ever been undertaken in any country. Based upon careful study of the best methods of foreign countries, and supported with generous appropriations and ample private contributions, which make possible the most complete equipment, this plan will follow the soldier through all the phases of re-education, find him employment or a market for his products, and insure sympathetic and intelligent co-operation from his family and from the community.

To carry out this program, a fortunate combination of public and private resources has been effected. Through the generosity of Mrs. T. Harrison Garrett, a suitable estate of ninety-nine acres at Baltimore was offered to the Surgeon General and accepted as the central hospital and school for blinded officers and men. The Red Cross War Council has supplemented the army's plan by creating the Red Cross Institute for the Blind, in order that certain phases of the social and economic supervision of these men may be provided for. The medical officer of the Surgeon General's staff, Lieut. Col. James Bordley, Jr., who is responsible for the official army program for the blind, has also been made director of the Red Cross Institute for the Blind, and will work in close touch with the Federal Board for Vocational Education, thus insuring thorogoing unity of policy and control.

This combination of an official organization with additional private resources will make possible the continuous and perpetual care of blinded soldiers after they are discharged from the army. Not only education and placement are contemplated, but subsequent industrial supervision for the balance of the soldier's life. It must be remembered that many blinded soldiers will, in addition, be suffering from other forms of disability. In blindness caused in the industries, 98 per cent of the eye injuries are without such complications, whereas in war 40 per cent are multiple; that is, these blind men also have amputations of arms or legs, or both, facial burns, destruction of jaws, and so on. To these particularly unfortunate individuals the nation owes a lasting duty. Patriotic zeal will provide adequate care for these men at present, but only invested capital and far-reaching plans will make their future certain.

As soon as the American soldiers, blinded in battle recover from their immediate wounds at the base hospital, the special work for them will be commenced. At first, the chief effort will be to amuse them and inspire them with some degree of hope. Later, as the men improve, they will be concentrated at the port of embarkation in France, under care of a special staff which will expend the measures of elementary instruction, teaching the men to get about and care for their immediate needs; in other words, teaching them "how to be blind."

On the way home they will be grouped according to the degree of injury, and upon arrival in the United States they will be sent to the United States Military General Hospital No. 7 at Baltimore for further medical and surgical treatment and special teaching. The technical training given by the Government will be the very best obtainable and only paid professional instructors will be permitted to give it. The idea of the Government will be to place every blinded man in a condition to take care of himself and those dependent upon him.

Where it is possible for a soldier to return to his old occupation, or one allied to it, the aim will be to have him do so. Otherwise a special suitable occupation will be taught, and in many cases it is hoped that the man will be able to command a larger salary after taking the training than before he lost his sight. Among the trades and professions which will be taught are broom-making, mattress-making, rug and carpet weaving, telephone operation, farm work, mat making, tuning in piano factories, winding of coils for armatures, piano tuning

as an individual trade, salesmanship, massage, typewriting and dictaphone operating. There will also be ample facilities for recreation in the school, including table games, dancing, singing, cross-country walking, roller skating, attendance at theatres, musicals, and other entertainments, medicine ball, push ball, punching bag, relay races, gymnastics, field sports, running, climbing, swimming, diving, and skating.

Some of the most interesting features of the general plan will be made possible by the supplemental Red Cross appropriation. The Red Cross Institute, for example, will undertake to provide a home and transportation to Baltimore for the relative who will be responsible for the care of the blinded man when returned to his home. It is the intention to instruct one such relative in the Government training school side by side with the blinded soldier, as is now done in the British and French armies. The purpose is that the blind man's family may be informed as to his difficulties, ambitions, training, and needs, so as to prevent the mental deterioration which often results from false sympathy. Friendly relations between the family and teachers will be a potent means of stimulating the interest of the blind man.

A reel of the moving picture about to be shown you will further emphasize the vocational side of the care given our blinded men.

The Division of Physical Reconstruction of the Surgeon General's office has as its chief Colonel Frank Billings, and one of the sections of this division has charge of the defects of hearing and speech under the directorship of Lieutenant Colonel Charles W. Richardson.

After many months of planning, on July 23, 1918, at General Hospital No. 11, Cape May, New Jersey, a school was established for the defects of hearing and speech in connection with this hospital. The plan of the school is that all defects of hearing and speech among invalided returning soldiers from the A. E. F., as well as those who have become affected in the cantonments, will be instructed or re-educated in this school before they are discharged from the army. In other words, all soldiers or sailors evidencing deafness or handicapped along the line of this work will, while having their physical disabilities corrected and cured, be re-educated and their handicap raised.

Returned soldiers and sailors having defects of hearing from whatever cause, either from disease or as the result of casualties, are those whose hearing is so reduced that they are handicapped socially and incapacitated industrially. They comprise all the completely deaf and those whose hearing is so reduced that it requires an effort for them to hear, and who experience difficulty in communicating through the spoken voice.

Corrective speech work will be carried out on all neuropathic affections of the voice, all types of paralysis (medical or surgical), and those in that large class of cases due to facial maxillary injuries.

The fundamental principle which has been adopted in connection with this service is to hold the men in the control of the United States Army as pupil patients until their physical handicap has been relieved. It has been found, in studying methods adopted by the various co-belligerents, that the retention of the soldier until his handicap is removed, especially for defects of hearing, is the only safe procedure. In the French and Italian armies, where this method is pursued, all men get their re-education before they are dismissed; therefore, all men with this disqualification or handicap are obliged to accept the type of treatment which is instituted.

In the British army where men presenting these conditions are discharged, it has been found that re-education is not taken up by 74 per cent. Indeed, all efforts have been made by the Aural Commission in Great Britain through propaganda, through entertainments, through the offer of meals, etc., to entice these men back to accept re-education. To the many who wish to work they have even offered evening classes, and yet they do not avail themselves of it, except to the extent of 24 per cent.

We feel, therefore, that centralizing these cases and holding them in the service is by far the safest, sanest, and most certain means to accomplish this purpose of re-education.

It is also well to remember, and we wish it placed strongly before the public, that these men whom we propose to re-educate are not abnormal, but simply have become handicapped through the loss of a special sense in their service to the common cause of liberty. It should also be understood by the laity that, through the methods which we propose to adopt in re-educating these men they will not be handicapped; that they will simply be required to read speech with their eyes instead of hearing speech with their ears. They will be able to take part in all social pleasures, and they will be able to follow almost all the gainful occupations.

In the section of defects of hearing we will use two principal methods in teaching these people. One is the auricular oral method, which, through using the voice and various types of electrical apparatus, bring into activity a sense which has been impaired, either through violence, "compression," or neuropsychic conditions.

The second method, and the one that we depend on to the greatest extent, is lip or speech reading.

In those very rare cases in which the individual is too obtuse, or is unable to concentrate, we shall be obliged to make use of the manual method.

Defects of speech arise from various complex causes, and comprise those cases which will have to be treated by neuropathic methods, those cases which will have to be met by respiratory methods, and those which will have to be taught the principles of phonetics. A large number of cases of face injuries involving the throat will not only have to be treated phonetically, but also surgically.

This work of physical and vocational reconstruction was started at Cape May under the immediate medical control of Major John M. Ingersoll, with Captains Berry and Lampe as assistants. These medical officers, under the Division of Surgery of the Head, will take care of all medical and surgical aspects of pupil-patients.

The educational department has as superintendent Mr. A. C. Manning of Mt. Airy, Miss Enfield Joiner of North Carolina as principal, and four teachers —Miss Mary Louise Wimsatt, Miss Margaret Bodycomb, Miss Clara Louise Rockwell, and Miss Mary Thornton.

The first class began on Wednesday, July 24th, 1918, with seventeen pupils, many of them completely deaf, most of them extremely hard of hearing. They form a bright, intelligent, enthusiastic group of pupils, ardent in their desire to learn, appreciative of the service being rendered them, and enthusiastic in regard to their prospects for complete rehabilitation.

NOTES, CASES AND INSTRUMENTS

AN EFFICIENT LAMP FOR EYE OPERATIONS.

F. H. VERHOEFF, M. D.

BOSTON.

Several years ago I was appointed chairman of a committee to provide satisfactory artificial illumination for eye operations at the Massachusetts Charitable Eye and Ear Infirmary. A previous committee had been unable to obtain on the market an entirely satisfactory lamp for the purpose, so I decided that it would be necessary to attempt to construct one. The lamp here described is the result of this attempt and has proved satisfactory in every way. It may be used to give either diffuse or focal illumination, but experience has shown that the former is seldom needed. The lamp consists essentially of a 6-8 volt nitrogen-tungsten automobile electric bulb, enclosed in a case, the light from which is focused by two convex lenses.

Fig. 1.

Fig. 1. Complete instrument showing convex lenses in place. (Verhoeff.)

Fig. 2.

Fig. 2. Lamp fixed on smaller cylinder ready for insertion in the larger. (Verhoeff.)

The case is an ordinary pasteboard mailing case, 5.8 cm. in diameter and 13.6 cm. in length. At a distance of about 3.8 cm. from the bottom, a hole is cut of proper size and shape to take a smaller mailing case, 4.2 cm. in diameter. A section 2.5 cm. long is cut from the open end of the smaller case, the threads for the screw top being left on. This section is glued into the hole in the larger case. The large case is now wrapped with adhesive plaster which is also carried around the smaller case so as to hold it more securely. See Fig. 1.

To the tin screw top of the large case is attached a third mailing case 4.2 cm. in diameter and cut off so as to be about 8 cm. long, as shown in the illustration. Fig. 2. This is done by first attaching a solid cylinder of wood about 2 cm. long (cut from a curtain rod) to the screw top by means of screws, and then sliding the cut end of the smaller case over it. A large hole to take the electric cord should be bored through the wooden cylinder and screw top. A hole is cut in the screw top of the smaller case, and an automobile lamp socket soldered into it. Seven or eight air holes are bored through the walls of the inner case, and four air holes through the screw top (not

the closed end) of the large case. No other air holes are necessary. The cases should now be given at least two coats of shellac inside and out. This is important, as otherwise moisture will be precipitated upon the lenses when the lamp is in use. To improve its appearance, the large case may be painted black or covered with leather.

The lenses consist of two trial case lenses with edges unground, each 41.5 mm. in diameter, one of 9 and the other of 18 diopters. These are kept from contact by means of a cardboard cylinder ½ cm. long. They are conveniently attached to the lamp by means of a threaded lens holder removed from a bull's eye flash lamp of suitable size. (See Fig. 1.) If this is not obtainable, a holder can be made of cardboard and attached by means of adhesive plaster. The stronger lens should be outside and at a distance of 5.5 cm. from the lamp filament. For diffuse illumination it is simply necessary to remove one of the lenses.

To obtain the proper current of 6-8 volts, we have made use of an electric generator connected with the house current, but a rheostat may be used instead. Sufficient current may also be obtained from a series of five dry cells of 1½ volts each. It is important that the electric cord attached to the lamp should be in good condition as otherwise sufficient current may not reach the lamp. The lamp never becomes too hot to be held in the hand. One of these lamps has been in use almost daily at this institution for over two years and is still in perfect condition.

QUININ AMBLYOPIA FROM RECTAL ADMINISTRATION.

C. S. G. Nagel, M. D.

SAN FRANCISCO, CAL.

Lieut. Col. R. H. Elliot's concise review of Quinin Poisoning (this Journal for August and September last) would seem to invite practical addenda. In the history I wish to submit "the drug was pressed, at a time when it should have been totally withheld," tho not for the reason assigned by Elliot (page 549).

On July 18, last, Mrs. G. H. aet. 60, presented herself with the following history. After an operation of colectomy for "fibroid of transverse colon" (apparently no anatomic examination) on June 17, 1917, another operation for gall-stones was performed a few months later on October 26. After the failure to inject something into the brachial vein during second day after operation, an (hypodermic?) injection was made over breast. The following day the patient received an enema of unknown character for heart failure, after which she fell unconscious for about thirty hours; and thereafter found herself totally blind for a month. She then began gradually to improve for several months, but had apparently made no further progress during the last few months. Both the patient and her husband stated that according to current rumor a mistake in regard to the enema had been made. (The reference to the few days between second operation and onset of blindness has always appealed to me as probably garbled, for natural reasons; and perhaps, also, others—this being a medico-legal case.)

Present Condition—R. Hm. +0.75=0.8. L. Hm. +0.25=0.6 with +3, Snellen 1 with some difficulty. Visual fields contracted concentrically close to point of fixation, except that in the left upper quadrant of each field there is a normal roundish area for white, and all colors to a maximum extent of 25° right. The fundus in both shows retinal arteries as well as veins very narrow, with absolutely white sheathing, more particularly of the arteries (thickening of walls). Both discs are of extreme pallor and sharply defined. Macula normal. The diagnosis of quinin amblyopia was made, with just a possible alternative of embolism of both central arteries (of which—simultaneously bilateral—there are four cases now on record), or rather remotely, ischemia retinae from loss of blood. After five weeks of strychnin treatment, vision had improved to 0.9 and kept the same under same treat-

ment up to the beginning of November. Visual fields had remained about stationary.

During my attendance the people had begun suit against the sanatorium where the operations had been done, and by the end of October a settlement was effected. The operating surgeon could now be prevailed upon to make the following statement. For the prevention of "post operative gas pains" he had ordered 5 grains of quinin hydrochlorid per enema, every four hours. After 30 grains (some always being lost, he figures on an actual intake of 25 grains) patient showed signs of failing heart. This was not attributed especially to the drug. However, the order was changed to every twelve hours. Instead the nurse administered the clysma every two hours. After 60 grains in all, the general condition of the patient becoming alarming (weak pulse, etc.) the drug was stopped. Loss of consciousness and blindness following, as related by the patient. No eye examination was made at any time during seven months; only a short while before I first saw her another oculist had examined her. Regarding the indication for quinin in this case, the doctor said that he had been very enthusiastic about it after an interview with another practitioner who claimed to have had extensive experience with the method. A leading surgeon confirms my suspicion that the method is unknown to surgeons in general.

I have failed so far to get the consent of the patient to try sclero-corneal trephining as proposed by Elliot. The procedure as defended by him appears worth trying. We know that in fresh cases of ischemia retinae a reduction of intraocular pressure thru iridectomy has yielded excellent results. This fact led me to advocate at the A. M. A. meeting of 1905 the early performance of iridectomy in methyl alcohol poisoning where I believe an ischemia retinae precedes the cumulative effect of formic acid on the eye. I now wish to go on record as recommending prompt broad iridectomy for fresh cases of quinin amblyopia, since contraction of retinal vessels here is so uniformly a primary feature, together with low general blood pressure and mostly normal intraocular tension.

OCULAR LESIONS OF INFLUENZA.

EDWARD STIEREN, M. D., F. A. C. S.

PITTSBURGH, PA.

From a paper read before the Allegheny County Medical Society.

The following observations were made during the present epidemic of influenza on patients admitted to the South Side Hospital from the beginning of the epidemic until December 10th, 1918, and from a smaller number seen in private practice.

Influenza, 221 cases; died 27. Mortality 12.2%.

Influenza with pneumonia, 104 cases; died 70. Mortality, 66.3%.

Influenza with pregnancy, 6 cases; died 6. Mortality 100%.

Of the total number of cases the eye department was called in to see 54, or about 16%. These were for the greater part acute congestion of the ocular conjunctiva, not a catarrhal type, but marked by a dilation of the bulbar conjunctival vessels with some edema of the conjunctiva, and not infrequently with small subconjunctival hemorrhages which soon became confluent. Photophobia was present in practically all these cases.

Cold moist compresses gave the greatest relief and usually resulted in an amelioration of the congestion in from 36 to 48 hours. In the few cases with a catarrhal discharge the palpebral conjunctiva was inflamed and rough, a simple collyrium of zinc sulphat and boric acid affording prompt relief.

A young man, convalescent for about a week, developed a violently inflamed eye, the palpebral conjunctiva, especially of the upper lid, taking on a finely granular or mulberry appearance. The cornea developed a uniform haze of the epithelium, so dense that the color of the iris could not be determined, vision being reduced to inabil-

ity to count the fingers at 6 inches. There was no pannus.

The application of various astringent remedies, 1% silver nitrat, alum stick, copper stick, even rubbing with 1-1500 bichlorid were without avail; dionin had no effect on the corneal haze and atropin was impotent in dilating the pupil.

Finally, calomel dusted into the eye proved to be the remedy needed, and the patient is now steadily making progress, the lids are almost normal and the corneal haze is limited to an area covering the center of the pupil, while atropin, instilled three times daily, has dilated the pupil to about one-half of maximum.

Repeated smears were made from the discharge from this eye, but at no time could any organism be detected.

Two cases of double cellulitis of the orbit occurred. Both recovered without the formation of abscess under constant moist Ochsner compresses.

Four of the six cases of pregnancy were examined with the ophthalmoscope shortly before death. The other two cases had died before it was realized how fatal influenza is to a pregnant woman.

All four had abnormally large pupils, which responded feebly to light stimulus. Two had a marked neuro-retinitis, (one of these with small flame-shaped hemorrhages), one had a fundus picture closely simulating an albuminuric retinitis; while the fourth had an extremely cyanotic retina, the latter case being complicated with pneumonia.

SOCIETY PROCEEDINGS

ROYAL SOCIETY OF MEDICINE, SECTION ON OPHTHALMOLOGY

November 6, 1918.

President, Mr. W. T. Holmes-Spicer.

The first meeting of the season was opened by the president's address.

Corneal Changes Following Inflammation.

The President first thanked the Section warmly for having elected him to its post of honor, and then proceeded to give an address, aided by numerous sketches shown by the epidiascope, on "Changes in the Cornea Which Follow Attacks of Various Forms of Inflammation." He said this subject had received but small attention in the literature.

After ophthalmia neonatorum, sometimes very little was to be seen but the existence of vascular channels, yet the stagnation of the lymph circulation might bring about slowly advancing changes after all the inflammatory appearances had long since subsided. The presence of clear lines in cases of old corneal ulcers had been described by but few observers, altho they had been long known, and had been seen before the introduction of the loupe. Nettleship described the linear paths of clear corneal tissue in large diffuse lipomata, the latter having probably arisen after small spots in infancy; while Fuchs discussed the various linear formations existing in the cornea, suggesting that the lines were due to changes in the lymph system and nebulous area. The best description extant of these lines was that by Sydney Stephenson. In one of his cases the lines traversed a nebula with opacity, and there had been repeated attacks of corneal inflammation early in life. Story stated he had seen lines of this character in interstitial keratitis, and he thought they were due to a clearing of vessels. The picture presented to the observer varied according to the depth of the vessels concerned. Besides the surface invasion of trunk vessels, the terminal capillary loops of the limbus were seen involved, these vessels not arising from any main trunk. This was common in marginal inflammations, such as trachoma and acute interstitial keratitis, in which vessels invaded the cornea at all depths.

It was quite uncommon for vessels to disappear and leave no trace; even though empty of blood, they could be seen as silky threads on the surface, or as clear lines beneath the surface. The vessels in one layer of the cornea did not interfuse with those in another layer. The first necessity in the production of these conditions was a destruction of tissue. The contraction which followed that either interfered with nutrition, or interrupted the flow of lymph, leading to stagnation and the deposit of salts. Arcus senilis was more likely to occur in eyes which have been the subjects of corneal ulceration.

The author then dealt in detail with the various forms met with.

On the motion of Mr. E. Treacher Collins, seconded by Mr. William Lang, the meeting accorded to the President a cordial vote of thanks for his address.

Dilatation of Retinal Veins and Arteries.

DR. HARLEY GOLDSMITH described and showed a case of excessive dilatation of retinal veins and arteries in one eye. When examined a few weeks ago for admission into the army, the surgeon said the vision of the affected eye was better than that of its fellow. The upper temporal vein was enormously congested, and the corresponding artery much dilated. Mr. Pooley showed before the Section, in 1909, a case of similar appearance which he regarded as angioma.

Nocardiasis of the Lacrimal Gland (Actinomycosis).

A paper by DR. J. B. CHRISTOPHERSON and MAJOR R. G. ARCHIBALD, of Khartoum, was read by Mr. Hepburn on "A Case of Primary Nocardiasis of the Lacrimal Gland, Caused by a Species of Nocardia Hitherto Undescribed."

The authors said the lacrimal gland appeared to be one of the most remarkable glands in the body. Having few disorders of its own, it fulfilled its destiny undisturbed by the disorders of the outside world, content to be left alone to administer its secretion to the great master tissue, the eye. The disease recorded in the paper represented nocardiasis, or actinomycosis, in which the causal agent was a germ or fungus, which grew readily aërobically and produced arthrospores.

The patient, a male aged 22, was admitted to the hospital suffering from a swelling of the right eye and inability to open it. The duration of the case was three and one-half years. There was neither scar nor wound visible. The eyelids could not be everted, but when raised a thick yellow discharge poured out, and the whole conjunctival surface of the upper lid appeared to be rough and granular, with deeper ulceration in parts. The swelling was painless, and was of dough consistence, tho without edema. There was a bulging forwards of the fornix of the conjunctiva. No lymphatic glands were enlarged, and the sight was unaffected. The eyeball itself was not invaded, nor were its movements involved; neither were photophobia or lacrimation present.

Under chloroform, the growth was dissected away. It appeared to have commenced in the lacrimal gland, and to have spread into the upper lid, invading the tarsal cartilage. It extended also somewhat between the eyeball and the bony orbit, along the ducts of the lacrimal gland.

Sections showed the morbid histology commonly associated with a fungal infection. A portion of a grain flattened between a slide and cover-glass showed that it was composed of the typical nocardial bacilliform hyphae, with rounded bodies or spores, the whole being held together in a dense matrix.

The paper then proceeded to describe the reactions in various culture media, and gave an account of the mycology. At the date of sending the paper the animal experimentation had not been concluded, but the condition did not seem to be pathogenic to the grey monkey.

Lieutenant Colonel R. H. Elliot said that tho actinomycosis was extraordinarily common in the endemic center of Madura and in Madras, yet during the many years he had practiced in the latter city he had not seen a case of it in the eye or any part of the orbit.

Mr. Leslie Paton expressed the cordial appreciation felt by the Section of the care taken by the authors to ensure the

safe arrival of the paper and specimens, especially as neither of the gentlemen was a member of the Section.

OPHTHALMOLOGICAL SECTION OF THE BALTIMORE CITY MEDICAL SOCIETY

November 20, 1918.

DR. FLEMING presiding.

Recurring Growth at Limbus.

DR. PEARCE showed a case of a peculiar epithelial growth of the limbus of the right eye, with the history that the patient had been under treatment several years; and that the eye had been operated upon three times for pterygium, with the recurrence of growth after removal. The cornea is now nearly completely covered with a peculiar leathery epithelial proliferation, a small portion of the cornea down and in still being free.

DISCUSSION: Dr. Crouch said that he recognized the patient as being one that he saw in 1911, at which time there was a small area covering the cornea at the temporal side and at that time he advised removal of a portion of the tissue for microscopic study, with the result that the patient disappeared from his observation.

Dr. Downey mentioned the fact that he had seen the patient for about two months recently, and that the epithelial extension had been very slow. It was the universal opinion that a portion should be removed for examination, and if malignant should have X-ray or radium treatment; and, if the process continued, enucleation of the eye.

Destruction of Lid.

DR. PEARCE also showed a case of rather extensive destruction of the lower lid in a woman of thirty, which, in the absence of Wassermann, was held to be a breaking down of a gumma.

Interstitial Keratitis.

DR. CLAPP showed a case of interstitial keratitis, which was brought before the Section to exemplify his opinion that some of the interstitial cases which do not improve under specific treatment are in all probability associated with a tubercular infection. This patient had been under treatment for many months with a very little improvement and specific treatment within the hospital was followed by very little change. The von Pirquet reaction was positive and the eyes have improved much more rapidly since the giving of tuberculin.

He also showed a case of Dr. Fleckenstein's which had been given tuberculin for two years, and the improvement had been very marked.

Carcinoma of Orbit.

He also showed microscopic sections of two carcinomas of the orbit, the first being a squamous cell epithelial growth starting in the bulbar conjunctiva, which did not improve under radium and later required exenteration of the orbit.

The other case was one of basal cell epithelioma which had been under radium treatment for five years, finally resulting in exenteration.

Sarcoma of Iris.

DR. FRIEDENWALD reported a case of sarcoma of the iris in a woman that he saw nine years ago for refration and later in 1915. In March, 1917, however, there was found a slightly pear-shaped pupil in the left eye, which upon examination with loupe showed a small growth at the nasal side of the pupil jutting out into the anterior chamber. Vision was 20/20; Wassermann reaction negative. The case was sent for consultation to Dr. de Schweintz, who confirmed his diagnosis of sarcoma. A very large iridectomy was done in May, 1917, and the tumor was sent to Dr. McCleary for examination, who reported spindle cell sarcoma. The vision has since remained fair with no recurrence to date.

C. A. CLAPP, *Secretary.*

ABSTRACTS

Lister, W. T.—Pathologic Aspects of Certain War Injuries of the Eye.

The Lancet, July 20, 1918. The writer presents a selection of observations made on certain war injuries of the eye which appeared either novel, or elucidated other allied conditions, or exhibited a contrast between civil and military experience. These are presented under five heads: 1. Ruptures of the sclera; 2. Concussion changes; 3. Suppurative changes; 4. Changes in the vitreous, and 5. Effect produced on retina and choroid by penetrating foreign bodies.

Regarding ruptures of the sclera: In civil life the greater number take place either immediately above, or above and internal to the cornea, and about 3 mm. from the limbus. In military practice, tho some are similar, others are quite different, and the cornea is burst forwards, or equatorially, or split into numerous petals. Many theories have been advanced to explain the site of civil ruptures, but none has received universal acceptance. Variations in thickness and strength of the sclera, and the amount of support derived from surrounding structures are important factors in determining the site of the rupture, but not the only ones, since otherwise all ruptures would take place in one zone. Lister points out that unless the sclera is actually perforated by the missile itself, rupture does not start from the point of impact, but occurs at some considerable distance from it. This is because the resistance of the envelope to pressure is one-third greater than its tensile strength; hence the eyeball would tend to burst at some distance from the point of impact, sooner than give way at this point.

Experience shows that ruptures most commonly take place either in the equatorial zone, about the line of impact, or opposite the point of contact (contrecoup), and it would appear that these two sites are associated with two different kinds of blows. I. The ruptures caused by slowly moving objects which strike the eyeball directly, without penetrating globe or orbit, occur in the equator round the line of impact at a point where the globe is least supported. This group will include the bulk of ruptures met with in civil life; they are caused by direct impact of comparatively slowly moving objects, such as stones, fists, sticks, boots, or cows' horns.

The site of impact is at the anterior half of the eye, usually below or down and out, owing to the protection afforded to other parts of the eye by the brow and nose; the line of impact from a blow below the cornea will pass from below upwards and backwards; the least supported portion of the equator to that line of impact will be above the cornea, viz., the site at which civil ruptures usually occur. II. In contrast to these we find that ruptures caused by rapidly moving missiles (to which the bulk of those seen in military practice belong) which pass through the orbit but do not strike the eye directly, occur at the side opposite to the point of impact—i. e., at the point of contrecoup; for example, missiles passing through the back of the orbit cause the cornea to be burst forwards, or if they have passed through the side of the orbit the rupture may be found near the equator on the opposite side. The writer explains in detail why the different forms of blows bring about different forms of rupture.

Next, the ruptures of the eye caused by a missile passing through the globe, are considered: When the foreign body passes through the *back* of the eye, the cornea is shot forward and the contents ejected, more or less of the cornea being torn away. When a sufficiently large fragment enters the eye in *front* there is an explosive effect, and the sclera is split into numerous petal-like lobes by radial ruptures extending backwards from the point of entrance. The elasticity of the sclera will accommodate the entry of small particles without rupture. When, however, the

fragment is of such a size and has such velocity that the united effect of the increase in volume caused by the entry of the body, together with the distending force of the pressure wave set up by its impact, is sufficient to overcome the elasticity of the supporting coat, widespread ruptures occur, splitting the sclera into leaves radiating backward, often close up to the optic nerve. This section of the article concludes with a comparison between ruptures of the sclera and fractures of the skull, tho the dissimilarities of the two structures prevent such a comparison from being very close.

The subject of some of the less common concussion changes is taken up. In discussing those of the retina and choroid it is pointed out that commotio retinae is frequently observed in military practice and does not differ from the civil type; several cases were seen where early pigmentation of the macula followed commotio, either as fine peppering or as somewhat diffuse greyish-brown spottings; a picture noticed, but not seen described, consisted of a peculiar striated appearance, evanescent in character, extending for several disc diameters round the macula, coming on when the white haze had disappeared and suggesting that the retina was still swollen and wrinkled. The more grossly concussed fundus met with in military practice, when unobscured by vitreous hemorrhage, is described as one of the most dramatic ophthalmoscopic pictures: In the early stages there are extensive, rich-red clouds of hemorrhage (retinal, choroidal and subhyaloid) interspersed with gleaming white patches; the latter gradually fade, and in the course of time are replaced by plaques of fibrous scar tissue, frequently festooned and sometimes perforated by round or oval holes; these plaques are probably patches of coagulation necrosis due to rupture of choroidal and retinal vessels in the immediate neighborhood, thus cutting off the blood supply and nutrition of certain areas of the retina.

The writer goes on to a consideration of the sites at which concussion changes occur—1, adjacent to the site of impact; 2, at the macula; and 3, immediately opposite the site of the impact. Missiles passing thru the side of the orbit may cause concussion changes at any of these situations; but blows on the front of the eye give rise to changes, as a rule, only at the posterior pole or around the macula. When missiles pass thru the orbit, the area always concussed is that adjacent to the site of the impact; so constant is this that in certain cases of wound of one side of the head, with no apparent disturbance on the other side with the exception of some blurring of vision, the ophthalmoscope by revealing concussion changes in the eye of this side will enable us to state with certainty, not only that a foreign body has passed across the head and thru the orbit on the opposite side to the wound of entrance, but also to define its course—a piece of information which may be of great value to the general surgeon. The macular concussion changes from blows on the front of the eye are common and to be anticipated as a result of bruising at the site of contrecoup; but such changes due to blows on the side of the eye are difficult to account for excepting that the greater vascularity and delicacy of structure in this region renders it more vulnerable; there may be two distinct patches separated by a band of normal retina. In some cases of a lateral blow on the eye, hemorrhages were seen immediately opposite the site of impact corresponding to contrecoup effects in the brain and sclera.

The effect of concussion on vision is discussed and such experiences summarized. In general, these correspond to those generally known. It is pointed out that in severe cases sight may be completely lost for a few days and may then be regained—a fact which may be of the greatest importance in deciding the fate of a wounded and concussed eye. Comparing the concussion changes of the fundus met with in civil and military life, the following striking difference is pointed out: In civil life, where concussion is due to blows of

direct violence on the anterior half of the eye from comparatively slowly moving objects, detachment of the retina and holes at the macula are comparatively common, while severe concussion changes are rare; in military practice, where the concussion is produced for the most part by rapidly moving missiles passing thru the orbit, detachment of the retina and holes at the macula are infrequent, while severe concussion changes are nearly constantly present.

Regarding suppurative changes in the eye, the writer says that, as would be expected from the large number of penetrating wounds of the eye in warfare, suppuration is common; but panophthalmitis, whether exogenous or endogenous, is usually not of an acutely infectious type; there is seldom much pain, and tenonitis with consequent proptosis and chemosis is not often marked. In endogenous or metastatic forms, Lister found much difference in the clinical picture according to whether the anterior or posterior vessels of the eye received the septic embolus. When the anterior half of the eye is first infected, there is early exudate into the anterior chamber and anterior portion of the vitreous and marked discoloration of the iris. When the choroidal or retinal vessels are the seat of the primary infection, the first sign is a general diffuse edema of the bulbar conjunctiva, loss of fundus reflex and dimness of vision, without discoloration of the iris or exudate in the anterior part of the eye; in the course of 24 hours there is added slight proptosis and practically complete loss of sight.

Next are described the changes which are produced in the vitreous, retina and choroid by penetrating foreign bodies; and finally the correlation of lesions with defects in the field of vision are discussed. Regarding the last, the writer says that the delicate localized lesions of the retina are in reality wonderfully neat physiologic experiments, for which in peace time it would be difficult to obtain either the subjects or the license. Experimenters have, by dividing portions of the retina in animals, been able to trace out the course of degenerated fibers in the optic nerve, but naturally, they were unable to correlate these lesions with defects in the field of vision in the lower animals; but in these war experiments on man we can correlate the visible lesions with defects in the field, tho we are unable to complete the examination by tracing the course of the degenerated fibers. Thus we find that lesions above or below the horizontal plane cause a defect in the field out of all proportion to the local disturbance—i. e., there is a "distribution defect," in addition to the local defect due to the lesion. This is due to the lesion having not only damaged the spot which is struck, but also involved nerve fibers which were passing on to a more peripheral part of the retina. The "distribution defect" is fan shaped from the point corresponding to the lesion and extending toward the periphery; the nearer the lesion is to the disc the greater the blind sector, and vice versa, which agrees with the accepted distribution of the nerve fibers in the retina.

When the lesion occurs in the horizontal plane there is no "distribution defect," but the loss of the field is limited to the area corresponding with the lesion, as would be expected, for the fibers supplying the retina in the horizontal line arch around from the disc to their destination, and therefore these lesions would only involve the nerve fibers at their termination.

Sector defects following bruising of the retina are strikingly similar to the sector defects in the field following solitary patches of choroiditis.

C. H. M.

H. K. de Haas.—Dangers from Arsenic in Daily Surroundings.—Geneeskundige Bladen, v. 20, No. 2.

This writer has found arsenic 32 times in the urine of sufferers with optic nerve inflammation of more or less specific character. Examining among his last 8,000 patients those who had optic nerve disease of a similar character, he had none with lead-intoxication, 4 with albuminuria, 3

with glycosuria and 22 with arsenicuria.

Comparing this with the reports of the ophthalmologic institutions in The Hague and Amsterdam he found the same percentage for albuminuric retinitis, while during that time no case was reported with lead intoxication or diabetic retinitis. Optic nerve affection and arsenicuria must be generally found in the Netherlands, and will appear more frequent when the facts are known. Ten of his cases had previously been seen by other ophthalmologists and he has the case histories of 5 more given him by colleagues.

The history of the first patient needs a rather full report. Mrs. —— came on July 3, 1907, with R. 3/60 and L. 6/6, hyperopia 4, 5 D. The amblyopia was connected with a right strabismus convergens. On March 2, 1912, she returned with complaints of difficult vision. This had gradually developed during the last months. The fundus was ophthalmoscopically normal, only below the left disc a spot in the retina seemed somewhat edematous. A few days later she complained of seeing spots and colors, suffered with a pronounced photophobia, and could not read or sew. The vision had not changed materially. The suspected spot in the retina was larger; showing in the direct image very fine, light, glistering points; also beaded capillaries. Here, therefore, was a retinitis, difficult to distinguish, situated in the superficial layers. The papilla had a normal color and border, only the veins were a little thick.

During the following days the process progressed along the course of the nerve fibers, and reached in some 2 weeks the macular region. Metamorphopsia was complained of, which showed that the process had gone deeper, reaching the layer of the rods and cones. "Everything seems to dance," relative and absolute, mostly negative scotomas and photopsies vexed the patient. During the following 3 weeks the retina regained its normal aspect, beginning at the disc, following the course of the nerve fibers in the direction of the macula, but not so regularly as the disease had done. No abnormality, objective or subjective, remained behind.

A scotoma corresponded with the diseased part of the retina, larger in the direction of the macula than the retinal lesion indicated. During some attacks the patient indicated scotomas, for which diseased retinal spots could not always be found, only a slight retinal hyperemia being seen. The patient's sturdy child remained always well and nursed. The urine was free from albumin and sugar. A tenia was present. The Wassermann was negative. The child was weaned, and the tenia destroyed. Looking for a cause of the intoxication much naphthalin was found in the linen in the bedroom; but its removal had no influence. The preceding summer the family had moved, the house had been painted and papered and floors broken open. Instead of "mouse-wheat," "rats-bane" was dusted about. On detection this was removed as well as possible. Was here a case of arsenic poisoning?

There were no symptoms of the conjunctiva, of the skin, respiratory tract or intestines, only some attacks of retinitis. Even before receiving K. I. she had a slight gingivitis, sometimes superficial pains in her face, sometimes intense dizzy spells, and rarely brief obscurations, during which all light perception would be absent. The urine for a week after the patient had been in the hospital contained arsenic, but so little, only a few thousandth parts of a milligram per liter, that de Haas did not consider that this could have any pathologic importance, especially as the examining chemist stated that he and his assistant did not have arsenic-free urine.

After a few weeks in the hospital the patient returned home free from complaints; but had a recurrence at the end of April; went away visiting, returned home cured at the end of May, and had another recurrence in June. The relapses were, like the first attack, superficial retinitis leaving the deeper layers intact. The cause must be in the house. These appearing and disappearing attacks were later somewhat explained.

During the next few years two events made arsenic still to be considered as cause of the retinitis, notwithstanding the examination of the urine wrongly considered negative. During a quiet intermittent spell the patient went to her dentist, who filled a tooth temporarily with arsenious acid. The same evening the patient had intense photophobia and only next morning had edematous spots, and very fine white foci in the eye. The same evening the dental canal was emptied, and the attack disappeared in a few days. The other attacks had ended more gradually, especially those where the patient remained at home.

Investigation showed that not more than 3 mg. of arsenious acid could have been put in the tooth. Looking for the cause the attention was attracted by the metal teapot. The patient stopped drinking all tea and coffee; and, as she had been advised to drink much, she consumed 1.25 liter of Vichy water daily. A sharp recurrence followed, on account of the two milligrams of arsenic which Vichy water contains per liter. The urine contained quite an amount of As. The air drawn from the locality where the paperhanger had strewn the ratsbane before the moving was found negative as to arsenic. But this was while the weather was good, and there was no moisture, so it was probably quite dry between that floor and the second story. At the end attention was drawn to the green painted wainscot in the kitchen, which was downstairs; this paint contained much arsenic.

Arsenic was also demonstrated biologically with a Penicillium culture. These fungi showed the possibility of evaporation of arsenic during moist weather; and also of their own presence at such times.

If it is true that chronic arsenical poisoning can be the result of combinations in gaseous form, formed by fungi from solid arsenical combination, thus may be explained the capriciousness of their appearance, and especially the stoppage of the attacks without the patient leaving the house. During moist weather, becoming warmer, when condensation appeared on the rather cold walls, the fungus throve and complaints came. During the dry weather and while it became colder, even if it rained, the fungus pined away, and the patient recuperated.

After removal of the paint, only one slight attack happened in the very rainy and mild month of January, 1916. No fire had been lit in the parlor so that we can safely surmise that it was moist there, and also in the ceiling above. After the fire was lit the attack disappeared. Vision is now as it was 10 years ago.

All cases had the following picture in common:

It was an affection of the peripheric optical conducting apparatus, limiting itself (as far as this is possible to determine clinically) to the most central neurons of the retina. The neurons were affected in the retina, or in the optic nerve, or in both. The lamina cribrosa did not form a barrier. Often the retina suffered first. Sometimes the retina did not show any distinguishable deviation. Then the changes of the disc alone were insufficient for differentiation from the classical intoxication amblyopias.

As far as objectively visible the inflammation was focal. This agreed with the functional disturbance, never being total blindness, remaining as local defects in the visual field. If the attack lasted long, fine pigment changes appeared in the pigment layer showing that the deeper retinal layers were affected. The light sense was little disturbed. Only a few patients, and they lightly, showed photophobia, which was so intense in the first case. The very fine superficial changes in the retina were only visible in the erect image. The retinal hemorrhages, when present were small, less than the diameter of an ordinary bloodvessel.

In at least eight cases, after removal from the arsenic source, an existing, previously stationary papillitis increased. In a retinitis four weeks, and even later after the ingestion of arsenic had stopped, appeared still papillitis. Under the same circumstances retinal hemorrhages (mostly only few,

situated near the papilla) would show.

The arsenical neuro-retinitis has mostly a continuous protracted course, lasting longer if the patients had been exposed longer. Many cases lasted 6 months or longer, after the ingestion of arsenic had stopped before recovery, or a permanent condition of atrophy. The central vision became damaged not only with retinitis, because this resided in and around the macula (sometimes exactly and only here); but also because the maculopapillar tracts of the nerve were first and mostly affected, even with the absence of any retinal changes. If atrophy was present the temporal quadrant was affected by preference. In serious cases the disc in its entirety became atrophic. In cases which had been long influenced by the intoxication, the vessels kept their normal caliber, unlike quinin intoxication. The visual field did not become contracted, but the para- and central scotomas, the permanent central blindness, made many needy persons. If vision became 1/10 it was only eccentric. *With few exceptions, the patients were otherwise healthy persons.*

Most of the cases could point to the wallpaper as the source of the intoxication. One soldered much, mostly tin. One, as a cooper, handled for many hours daily galvanized iron hoops, in the tin of which was much arsenic. Many became affected in an institution where the lids on the pans in which the meals were prepared, were made of galvanized steel; that is sheetiron which was not tinned over but covered with zinc. This contains regularly arsenic. Here and there the zinc was absent. Could it have been dissolved in the food? Investigation showed that the institute inhabitants absorbed less than 200 mgrm. of As_2O_3 daily; that is such a quantity of arsenic that it becomes after oxidation 200 mgrm. As_2O_3. This quantity of arsenic at the utmost, combined in unknown form, in which it reaches daily the intestinal wall with the food, made two persons lose their central vision.

Of the 13 people examined in the institution, 2 had an arsenic-free urine, altho undoubtedly arsenic was absorbed. Not every one's kidneys have the same permeability for arsenic. The seriousness of the ocular manifestations did not depend on the quantity of arsenic found in the urine. Only a few of the many hundreds, living in the same conditions, showed ocular disease. None of the other institution physicians had found the slightest indication of arsenic poisoning during the last year. The only two who received permanent damage, were two sisters. Zinc kitchen utensils are dangerous, unless the zinc proves on examination to be free from arsenic.

One of the 35 patients, a boy of 18 years, when last seen had still 10 mgrm. of arsenic in his urine. He could count fingers at 4 meters with R. He could not see more until of 5/20 he saw well one letter, of 5/15 two letters and of 5/10 he read them all correctly (E T L F N). He had with the same eye less than 5/60 and more than 5/10 vision. Somebody who tumbles over a pail, which he does not see, while he is able to pick up a pin, becomes almost suspected of simulation. This is the result of central sharply limited defects in the visual field.

De Haas could find in the literature 8 cases of arsenic optic nerve poisoning; 6 thru administration of arsenic as medicin, or as a poisoning dose by the mouth, and two thru wall coverings. Arsenic poisoning of the optic nerve has been described rarely up to date, except thru atoxyl, arsacetin, etc.

The normal urine does not contain arsenic. The urine can contain arsenic in clinically healthy persons; when they live in surroundings containing large amounts of it.

Of the 35 cases there were 32 where De Haas made the clinical diagnosis of neuroretinitis arsenicalis, and arsenic was found in the urine in which the quantity did not amount to more than 60 mgrm. per liter urine. The three exceptions could be readily accounted for.

The consequences of the loss of vision thru avoidable arsenic in the practice of de Haas alone represent a value of a million guilders. E. E. B.

MacNaughton-Jones, M.—The Sterilization of Local Anesthetics.—The Lancet, June 29, 1918.)

The writer suggests a simple but effective method of preparing a sterile solution of cocain or other local anesthetic which may also contain adrenalin. It consists in preparing a solution of sodium chlorid together with the anesthetic or anesthetics and adrenalin, each constituent being present in such quantity that a dilution with many (e. g., 40) times the volume of water, will give a solution containing the requisite percentage of each. The preparation is placed in a sealed phial or ampoule and kept until required. Sterile water is added immediately before injection. Experiments demonstrated that even if the preparation be inoculated with the ordinary forms of bacterial contamination the presence of concentrated salt renders it sterile in a few days.

A simple method of preparing the concentrated solution is to take the quantities of anesthetic, salt, etc., required per 1,000 and dissolve in water; but make up to 25 instead of to 1,000. The concentrated solution suggested contains:

Kerocain or procain (novocain)	3.25	(13%)
NaCl	3.25	(13%)
Adrenalin chlorid solution (1 in 1,000)	8.00	(32%)
Water by weight (or metric)	ad 25.0	(100)

This solution, prepared without antiseptic precautions, and after contamination with bacillus coli and other organisms, was sterile a week after preparation. When diluted with 40 times its bulk of water it contains 3.25 parts per 1,000 of both procain and sodium chlorid, gives perfect anesthesia, and has an osmotic pressure approximately equal to that of a 4 per 1,000 (0.4 per cent), solution of the latter salt. The adrenalin is not in sufficient strength to arrest hemorrhage, but secures prolonged anesthesia. A certain amount of hemorrhage during the majority of operations is easily controlled; and, in his opinion, is preferable to the risk of a subsequent hematoma, which he has seen develop on more than one occasion.

If a 2 in 1,000 (0.2 per cent) solution of cocain be desired the formula would be modified as follows, and the concentrated solution would be:

Cocain hyd.	2.	(8%)
Sodium chlorid (approx.)	3.75	(15%)
Adrenalin chlorid (1 in 1,000)	6.	(24%)
Water by weight to	25.	(100)

It is suggested that a solution be prepared 40 times the strength of that required for injection, because increments in the dilute solution of ¼, ½, ¾ or 1 per 1,000 correspond to an increase of 1, 2, 3, or 4 per cent. in the concentrated solution, and these are the increments usually desired.

C. H. M.

Magitot.—Traumatic Variations of Ocular Tension.—(Ann. d'Oculist. v. 155, p. 1). This author refers to a previous work in which he proved that ocular tension was dependent on the amount of blood in the uveal vessels. (See A. J. O., v. 1, p. 587.) The aqueous is a practically stagnant fluid permeating the fibrillar neurogleic sponge called the vitreous. Being incompressible, it gives way when the erectile choroid tissue becomes distended, and becomes richer in albuminous contents as the result of serum passing thru the vessel walls.

Therefore the permeability of the iris angle and the canal of Schlemm has no influence on ocular tension. If the aqueous were concerned in tension, an increase in the contents of the eyeball caused by a hemorrhage into the anterior chamber should be followed by a rise of tension instead of the fall which actually occurs. The regulation of the blood in the uvea is under the control of the intraparietal nerve cells of the eyeball, at least they are without doubt the point of origin of the reflex caused by the stimulus of the trauma, which results in an excitation or inhibition, with consequent hyperemia or anemia of the choroid.

Traumatic lesions of the eyes can be divided into two groups: (a) where the lesion of the orbit predominates,

and (b) where the ocular lesion is the more important. The first group consists chiefly of hemorrhage into the tissues, which interferes more or less with the venous flow. However, it does not completely check it, and moreover there is an immediate beginning of absorption which causes a progressive decrease in the interference. In three cases which he quotes there was a decided rise in tension following interference with the venous outflow, altho the eyeball was uninjured. In a case of unilateral anthracoid furuncle of the lid with orbital phlebitis the tension on the affected side was 45 mm. and on the healthy side 15. The vision was normal, however, there were no fundal changes, the pupils were equal, the tension fell in the same ratio as the inflammation subsided and the stasis was relieved.

In group (b), characterized by the predominance of ocular symptoms as opposed to orbital, there may be either a rise or a fall of tension. In some cases there is an inhibition of the intraparietal nerve cell, resulting in a passive dilatation of the arterioles, an overfilling of the veins of the uvea with a turgescence of the latter and a rise of tension. In other cases there is an excitation of these cells, a contraction of the arterioles, less blood in the veins, shrinking of the uvea and fall of tension. It is impossible to say why the trauma has one effect one time and another the other, for it is impossible to point out the exact spot where the trauma may be acting. Then, too, a moderate effect may stimulate and a more severe one may inhibit the physiologic action of the same cells. In a case of ophthalmomalacia very striking oscillations of tension were noticed.

In considering the disease glaucoma, the error is frequently made of confusing the symptom hypertension with the disease itself. As a matter of fact, there are many ocular affections, some inflammatory, some angiosclerotic, some purely traumatic, which, according to the point of attack of their etiologic agent on the vascular element of the uvea, or its innervation, may cause a high or low, transient or permanent tension.

His conclusions may be summed up as follows:

Traumatic variations of ocular tension have two causes—one passive and the other active.

1. Mechanical interference with the orbital venous circulation. These hypertensions last as long as the circulatory obstruction is present.

2. Troubles caused by the action of the trauma upon the intraocular vasomotor nervous system. C. L.

Sakai.—Sensitiveness of the Cornea and the Conjunctiva.—Nippon Gank. Zasshi. Dec., 1917.

The results of his experiments follow:

1. The conjunctiva has the sense of touch as shown by Nagel, but less, relatively than the edges of the lids, skin of the face, etc.

2. The bulbar conjunctiva on the part exposed by the aperture of the lids has a little less sensitiveness than the other parts.

3. The cornea has the sensation of touch.

4. The cornea and conjunctiva fail to have the sensation of warmth, and this is likewise so of the caruncle and semilunar fold.

5. The bearable heat is 42-43° (Cent.).

6. Pain is especially experienced in the cornea.

7. It is in spots in the conjunctiva especially in the lids, more than that of the bulb. This condition may be observed also in the cornea (some portions being more sensitive than others).

8. The difference between the sensitive points is the least on the free border of the lid (8.7 mm.), while that of the conjunctiva is 15 mm.

9. Sensation to chemical substances is less than that to cold.

KOMOTO and H. V. W.

Wallis, Hoare, W.—Operative Technic and After Treatment of Cataract Extraction.—(The Medical Journal of Australia, 1918, p. 297.)

The writer says he ignores the ques-

tion of ripeness, and operates on the riper eye if the patient cannot carry on. After making a conjunctival flap he does a large ring-shaped capsulotomy and a peripheral iridectomy. Twenty-five per cent argyrol solution is instilled for two days before and the conjunctival sac irrigated with normal saline, just before operation. He contents himself with this provided a culture shows no danger from the conjunctiva. He lays great stress on the importance of retraction of the lids, uper and lower, by an assistant using Lang's or Harman's retractors and pulling forward and away from the globe (Smith). The loss of vitreous he looks upon as comparable to shock, hemorrhage or sepsis in general surgery. Intraocular irrigation with saline after extraction and eserin are used. A sterile vaselin swab is applied and held in place by plaster and a wire mask. Aged patients are out of bed in two days. A large number, irrespective of the type of operation, have to undergo discission.

F. P. M.

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GERMAN OPHTHALMOLOGY.

A myth is sometimes worthy of careful consideration and scientific study. There has been an attitude of exaggerated appreciation on the part of Americans toward Germany, and its supposed achievements in ophthalmology, so out of proportion to the real share Germany has taken in the development of ophthalmology, that it is worth examination. The power to inspire confidence and unmerited respect is an attribute of the successful quack the world over. The disposition to use propaganda to advance the interests of "kultur" is a German characteristic. At this time of careful sifting it is well to inquire how far the respect shown for the ophthalmology promulgated in Germany is justified by real scientific leadership; and how far it is based on unthinking acceptance of an assumption of superiority that does not exist.

The expansion of knowledge, interest, and achievement which characterizes modern ophthalmology, began in the middle of the last century with the use of the ophthalmoscope, the exact measurement and correction of anomalies of refraction, and specialization in practice. The use of the ophthalmoscope was given to the ophthalmologist largely by German workers. When Helmholz had solved the problems of physics involved in seeing the background of the living eye, Graefe, Jaeger, and their colleagues took the instrument and began making studies of the eye-ground. Babbage had invented such an instrument ten years earlier, but the English surgeon to whom he showed it failed to perceive its importance; and it was put aside unused, until the credit of its introduction had been secured for Helmholtz and Germany.

But the important practical improvements that have been made in the ophthalmoscope have nearly all been suggested and applied outside of Germany. Reute of Holland gave the perforated mirror, which Wadsworth of Boston and Loring of New York perfected in the tilted form. The socalled Rekoss disc was of little importance until the New York optician, Hunter, working for Loring, Knapp, and John Green, had perfected their forms of refraction ophthalmoscope. Morton of London suggested the other widely used mech-

anism for a practical lens series. The electric ophthalmoscope was suggested and perfected in America by Dennett, Marple, and their successors. The latest advance, in the large fixed ophthalmoscope, has been made by Gullstrand of Sweden.

The most important offshoot from ophthalmoscopy, the shadow test, skiascopy, has been wholly developed and principally used outside of Germany. It began with incidental observations by Bowman, was developed into a method by Cuignet, Mengin, and Chibret; and has been perfected by French, English, and American workers. Transillumination, first described in Germany, remained as little noticed and as unused as the Babbage ophthalmoscope for 30 years, until taken up by Swanzy of Dublin, and Würdemann and others in America.

The ophthalmometer, a ponderous laboratory instrument of Germany, that has measured the eyes of probably less than 100 persons in any generation, was first introduced as an instrument for clinical work, when Javal and Schiötz had based it on new principles and remodeled it. Since then it has been used on hundreds of thousands of eyes, in the practical measurement of refraction.

The scientific understanding, exact diagnosis, and practical correction of errors of refraction began and developed outside of Germany. From the time of Young, Airy, and Stokes of England; of Donders, whose work in Holland, translated from the Dutch in Dublin, first received adequate publication in the new Sydenham Series in London; thru Landolt, Weir Mitchell, Thomson, Green, Dyer, Norris, Hay, Risley, Brudenell Carter, Hartridge, and Gould, the correction of ametropia developed to more than half of the work of the ophthalmologist, without ever being well taught or its importance fully understood in myopic Germany.

Operative ophthalmology comes down to us from an older time, and each generation modifies and readapts the older procedures. Cataract extraction had, of course, been introduced by Daviel before any German school of ophthalmology existed. The generation that followed him devised and used knives of all possible shapes, that of Beer remaining most generally popular. Graefe, choosing a narrower knife, such as had been used by Sharp, described what he called a modified linear extraction; which no one now practices, and which he soon modified himself. This, with a few such suggestions as Waldau's spoon, modified by Graefe, but properly supplanted by the Levis wire loop, constitute the chief original contributions of Germany to cataract extraction.

After Chevalier Taylor had claimed to do an operation for squint, and Bell had suggested a rational basis for it; Stromeyer worked out an operative procedure, and Dieffenbach of Berlin did this operation and exploited it to the world. Dieffenbach probably did it after Guerin of Paris; but he got into the newspapers with it first; and then every general surgeon from Moscow to Peru began to use and modify it. Dieffenbach's record of 3,000 cases in three years has probably never been broken. But so recklessly was the operation resorted to, and so bad were the ultimate results, that it quickly fell into complete disrepute. So when Graefe began to publish accounts of practically the same operation 15 years later, it was mentioned as Graefe's operation. Revivals of the squint operation brought about by Critchett and Snellen were better procedures; and much more in the line of modern progress than the Graefe operation. The muscle operations for advancement, tucking, and resection have all been developed outside of Germany. From German operators we have had only inferior elaborations or imitations, not even a new method of placing or tying the sutures.

The great advances of modern general surgery began with Lister's application of the discoveries of Pasteur. Sir Berkeley Moynihan has brilliantly shown how little Germany has contributed to this movement. In plastic surgery about the eyes, Germany has contributed less than England, France, Italy or even India; and now Amer-

ica's practical inventive ingenuity, turned to the solving of its problems, is achieving unprecedentedly good results in blepharoplasty and restoration of the socket. In the surgery of adjoining parts, of the brain, the sinuses, the hypophysis, America has taken the lead. West, of Baltimore, taught his window resection of the lacrimal passages in Berlin, apparently understanding that German surgeons and their students and imitators would never recognize its merits if taught anywhere else.

In view of the limited proportion of the advances in ophthalmology that can be traced to German sources, what are the factors that have entered into the making of the myth of German ophthalmology? A study of them can be of special practical value to American ophthalmologists today. The myth has been founded chiefly upon the statements and claims of Americans who went to Germany to study ophthalmology; and came home to impress the medical profession and the public with the mastery of it they had attained there.

When modern ophthalmology began to develop as a medical specialty, every medical school in America was a private business enterprise, dominated by general practitioners and surgeons who were strongly opposed to specialization in medicine. There was no reputable, well-educated physician in America formally attempting to teach ophthalmology. All that it was generally considered necessary to know about the eye and its diseases, was taught by the professor of general surgery in two lectures.

Under these conditions young men like Williams, Dyer, Green, Holmes, Noyes, and Agnew, who became interested in the subject, went to Europe to study it, under Bowman, Desmarres, Donders, Graefe, and Jaeger. Graefe himself had studied in Paris and London, then world centers for ophthalmology. Naturally the younger men who caught their interest in ophthalmology from its first teachers in America, as Wadsworth, the Derbys, Loring, Norris, Thompson, and others, turned to the springs of knowledge that had been so much to their teachers. A still younger generation found in Germany the laboratory opportunities that were still scarce everywhere, and a few utilized them.

The idea of finishing an education for ophthalmic practice by a year, more or less, in Europe became firmly implanted in the mind of the American medical profession. Germany offered certain conditions that were especially attractive to the student who had been biased with the notion that all he needed was clinical contact with cases of eye disease to make him an ophthalmologist. People raised for "cannon-fodder" can easily be utilized as "clinical material." From masses, compelled to live under conditions of poverty and social degradation could be built up public clinics of such size and subserviency as were impossible anywhere else in the world.

To the claims of these big ophthalmic clinics the American medical student was peculiarly susceptible. To see six cases of retinitis pigmentosa in one day, seemed a vast clinical opportunity. In his ignorance of real clinical study he did not realize that he could have learned a great deal more from one case studied for six days. In the last quarter of the nineteenth century, Vienna and Berlin had come to be regarded in America as clinical Meccas; and the priests of this religion lived by the fees of American students.

To have gone abroad to "study" served the purpose of impressing prospective patients; and few were capable of judging the character of that study or the value of its results. German ophthalmology including a superficial clinical acquaintance with the common diseases of the eye, a contempt for the careful measurement of ametropia, and an exaggerated desire to discover rare cases, became the popular standard of attainment for the eye specialist in America. More practical French and British clinical work, and the American passion for results, were needed to save ophthalmology from becoming a mixture of clinical superficiality and laboratory pottering.

When it became understood in German clinics that ophthalmology was unknown as a medical specialty in America, a very natural emigration of German ophthalmologists occurred. Julius Homberger was one of the first. But later came men like Knapp, Hotz and Alt, of scientific attainments worthy of the respect they received from their American confreres; and it happened that the first three ophthalmic journals published in this country were started by men who had been born and educated in Germany. Moreover, the first American institution for graduate teaching in ophthalmology was Knapp's Ophthalmic and Aural Institute in New York.

The student commonly has an exaggerated estimate of the knowledge and wisdom of his instructor. He ranks high the man who taught him, and regards him as an authority. Whether this feeling was exploited in the clinics, or in the literature of Germany, it produced a respect for German achievements in the field of ophthalmology out of all proportion to their scientific importance, a readiness to accept anything that came thru German channels, while important communications from other sources were unnoticed.

It seems to be natural for the German scientist to spend his time in the recording of minute routine observations, and in collecting bibliographies. There grew in Germany a mass of literature, some of which was valuable, and some merely cumbersome. Mackenzie had done much for ophthalmology in this direction; but in "Nagel's Jahrsbericht" and in elaborate bibliographies the Germans brought together all the papers related to each single topic. Their ophthalmic literature was often ponderous, sterile, and marked by sordid, bitter controversies on questions of priority. But its mass made an impression on those who did not read it; and students were able to search out in it, that which had not been gathered or preserved anywhere else. It helped to give plausibility to the myth of German ophthalmology; and substance to the commercial returns that myth was capable of yielding.

With ophthalmology universally recognized as an important branch of medical knowledge, and practice; with schools and literature developing in all parts of the world, the unreasonable reverence for German ophthalmology was doomed. But it is useful to know that its influence rested on two things: the establishment of institutions where ophthalmology was taught, and the development of a literature in which published facts were carefully preserved and collated. The comparative value of such institutions and this kind of literature need to be better recognized in America. When they are so recognized, we will develop better teaching and a more valuable literature than those which helped furnish prestige to Germany. E. J.

SILVER PREPARATIONS IN LACRIMAL SAC DISEASE.

Even yet, despite certain unpleasant medico-legal experiences of some American oculists, do we find many men using the silver solutions as the nitrat, argyrol, and protargol for injection into the lacrimal sac for dacryocystitis.

Now, the best of us abrade the lacrimal passage mucous membrane, and even occasionally make a false passage in sounding. Then too, in quite a proportion of such cases erosions have already occurred in the course of the disease; and the forcible injection of any fluid by the syringe may carry the solution into the tissues of the lid or orbit.

If this fluid be aseptic and colorless no great discomfort may ensue; but if such a substance as silver is used, which makes an indelible stain after exposure to light, permanent discoloration of the lower lid—a chronic black eye—may occur.

This, in the case of a young person, especially in a good looking woman, is a great disfigurement, which can not be removed by any known treatment. Certainly the courts have upheld the charge of malpractice in such cases.

Reiterated warnings are necessary regarding these solutions, for their use is common in this connection.

H. V. W.

POST-WAR AVIATION

Whatever else may happen as a result of the war, one thing is certain, namely, that the knowledge acquired concerning the mechanics of aircrafts, and the training of thousands of aviators will bring it to pass that air transportation will soon aid, possibly rival, land and sea transportation. This will happen either as government controlled enterprises, or as large corporations similar to the present railroad and steamship companies or as individual action similar to the present use of automobiles. It requires no great exercise of the imagination to see the air filled with crafts devoted to sport or profit. Also, it is evident that if this occurs, in the course of time regulations concerning their conduct will become necessary. Will the world wait until the problem becomes complex, or will some far seeing statesman initiate these regulations now, before vested rights and customs have arisen to demand consideration? Will they be municipal, state, national or international?

It is not the province of this journal to discuss the question as to what level shall be occupied by each form of aircraft, and what the "rules of the road" shall be, nor any of the other regulations of similar character, but it is our privilege to call attention to one phase of the question, and that is the *medical* qualifications of the aviator. Two extremes are possible; one is to follow the custom that obtains in the case of automobiles, and permit any one or any age, physical condition and habits to become an aviator. The other is to follow the rigid requirements of the army aviation service. Of the two, the latter is by far to be preferred, but is there not the possibility of adopting less stringent regulations without sacrificing safety. For example, is 20/20 vision without glasses essential for peace time aviation? How far should correct color vision enter into the requirements? Is correct response to the equilibrium tests equally necessary for those who fly at low altitudes as those who fly at high? Should alcoholics and people of unstable mental states be allowed to fly at all? These and many other problems should be settled now, when flying is in its infancy. It will be easier to exclude unfit flyers before they start flying than afterwards. On the other hand, the enforcement of unjust regulations might keep many men from flying who really ought to be allowed that privilege.

It seems to the writer that the best way of settling the medical problems connected with the future of aviation is for the large, representative medical organizations—the American Medical Association, etc.—and the special ones in whose province these questions lie—the Ophthalmologic and the Otologic Societies—should appoint committees to study them, either separately or conjointly, and make recommendations to Congress, so that the regulations when made shall apply equally to all men and all parts of the country. Let us hope that the first of these to meet in 1919 will initiate the movement, and that the others will join in, so that we may have scientific and not haphazard regulation of what promises to be one of the greatest fields of human activity.

C. L.

BOOK NOTICES.

PHYSIOLOGIC OPTICS, by **Charles Sheard,** A. B., A. M., Ph. D., Professor and Director of Applied Optics in the Ohio State University. Reprinted from the American Encyclopedia of Ophthalmology. 8vo. 517 pages. 250 illustrations. Cloth. Price $5.00. Chicago, Cleveland Press, 1918.

Deficiencies in their training in physiologic optics are felt by many American ophthalmologists. Improved methods and opportunities for study of this branch are among the most urgent needs of our courses of preparation for ophthalmic practice. The most com-

plete book on the subject hitherto available in English, has been the book by Tscherning as translated by Weiland 19 years ago. Of Helmholtz work there was no English translation. As a student, the writer regretted this, for the German text was difficult and forbidding. He has sometimes wondered whether the absence of a good textbook has had something to do with the neglect of the subject by many ophthalmologists in America.

However that may be, from the time of Thomas Young, many of the most important original contributions on the subject have appeared in English. Now, in the work of Sheard, we have a treatise that beside doing justice to the facts and formulas gathered and developed by Helmholtz, brings together the large number of practical observations and propositions recorded by more recent writers, especially in English.

It is fortunate that, as it originally appeared in the American Encyclopedia of Ophthalmology, it is already at the command of the large number of subscribers for that work. It is also well to have it available to those who have not the Encyclopedia, by publication of this separate volume.

In any treatise on physiologic optics, the algebraic formulas that are so noticeable are likely to deter some ophthalmologists from reading it; and prove a real obstacle to those who do undertake its perusal. It is but fair to call attention to the fact that in this work many of the laws that are commonly expressed by such formulas, are also stated in clear, direct English, or are made clear by simple, well chosen diagrams.

It should be realized also that physiologic optics is a much broader subject than mathematic optics; and that many things here set forth need no mathematics to explain them. This becomes evident when we examine the table of contents.

Part I. Ocular Optics, including refraction at curved surfaces and catoptics, gives full scope to the methods of mathematics; but the other parts have much less use for them.

Part II. Monocular Vision, and the methods of objectively and subjectively investigating it, is less formidable and of greater practical interest. The same is true of the subsequent parts.

Part III. Qualitative and Quantitative Determinations of the Retinal Functions.

Part IV. Binocular Vision and Ocular Movements; and Part V, Binocular Vision and Disorders of the Motility. In these latter parts there is much of interest to every one engaged in ophthalmic practice; and little that will make an excessive demand on his preliminary training in mathematics.

It is not possible in this notice to indicate the many subjects discussed in this book, that must interest any one who wishes to understand the eye and its functions. It must suffice to state that it covers physiologic optics as that branch of science has developed up to date. Of the four brief appendices, not included in the original article as published in the Encyclopedia, one is devoted to "The Theory of Color Vision," as reviewed by Mrs. Ladd-Franklin; another to "Refractive Differences in Foveal and Parafoveal Vision;" and a third on "Stereoscopic Colors or Retiring and Advancing Colors;" all based on articles published within the year.

While ample justice has been done to the workers of other countries, those of America have not been slighted, while Maddox is referred to 14 times, Howe is mentioned 12, Stevens 14, and Savage 19; and Cobb, Ferree, Ives, Lippincott, Luckiesh, Nutting, Prentice, Weymouth, and others have their work adequately noticed. Among the American writings regarding physiologic optics may be found ideas that will not be permanently held; but even these compare favorably with the errors of the past, and all that are included in this work are worthy of study and consideration.

On the whole, this book is excellently written in clear, direct language. It contains a better and more complete account of its subject than any other treatise yet published in any language.

It is well illustrated, neatly printed, and has good indices both of subjects and names, beside the full table of contents. The appendices and indices, the principal additions made to the work as it appeared in the Encyclopedia, add much to the value and convenience of the book as a work of reference. E. J.

TRANSACTIONS OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA, Third Series, Volume 39. 582 pages. Illustrated. Printed for the College.

This volume covers the transactions of the College for the year 1917. The 18 months that have elapsed since the publication of its immediate predecessor, illustrates the difficulty of getting high grade printing done under the conditions existing during the last year. Its principal interest to the ophthalmologist lies in the 59 pages devoted to the proceedings of the section on ophthalmology. These proceedings have already been published more than a year ago in the Journals which were combined to form the AMERICAN JOURNAL OF OPHTHALMOLOGY. The volume is a very creditable one and contains a number of important papers and discussions of general interest to all members of the medical profession.

Among these are communications on "The Significance of Spinal Fluid Findings," by Fordyce; and upon "Syphilis of the Central Nervous System," by Swift and Stewart.

TRANSACTIONS OF THE THIRD COLORADO OPHTHALMOLOGICAL CONGRESS, held in Denver, 1917. 59 pages. Illustrated.

The papers included in these transactions have already been published in the AMERICAN JOURNAL OF OPHTHALMOLOGY, and are reprinted from this Journal chiefly for circulation among the members of the Congress. E. J.

NEWS ITEMS

Personals and items of interest should be sent to Dr. Melville Black, 424 Metropolitan Building, Denver, Colorado. They should be sent in by the 25th of the month. The following gentlemen have consented to supply the news from their respective sections: Dr. Edmond E. Blaauw, Buffalo; Dr. H. Alexander Brown, San Francisco; Dr. V. A. Chapman, Milwaukee; Dr. Robert Fagin, Memphis; Dr. M. Feingold, New Orleans; Dr. Wm. F. Hardy, St. Louis; Dr. Geo. F. Keiper, LaFayette, Indiana; Dr. Geo. H. Kress, Los Angeles; Dr. W. H. Lowell, Boston; Dr. Pacheco Luna, Guatemala City, Central America; Dr. Wm. R. Murray, Minneapolis; Dr. G. Oram Ring, Philadelphia; Dr. Chas. P. Small, Chicago; Dr. John E. Virden, New York City. Volunteers are needed in other localities.

DEATHS.

Paul Chavernac, of Marseilles, was killed recently in active service.

A. H. P. Dawney of London, died November 8th, of pneumonia; aged forty-eight years.

Isaac H. Lent, Middleton, New York, aged seventy-two, a member of the Medical Society of the State of New York; consulting ophthalmologist to the Thrall Hospital, Middleton; died at his home, about October 20th.

Dr. Albert B. Mason, Atlanta, Ga., aged thirty-one, died at his home, December 4th, from pneumonia following influenza.

Dr. Wm. H. Moore, Martinsdale, Mont., aged forty, died at his home, December 1st, from pneumonia.

Dr. Thomas T. Perkins, Cliftondale, Mass., aged forty-five, died at his home December 6th, from heart disease.

Dr. Eugene V. Riker, Flint, Mich., aged fifty-seven, died at his home, November 25th, from pneumonia.

Dr. Glenn C. Spurgeon, Seattle, aged forty-four, a member of the staff of the Eye, Ear, Nose and Throat Infirmary, died at his home, October 29th, from pneumonia following influenza.

Capt. Halstead Robert Wright, M. C., U. S. A., Columbus, Ohio, aged forty-three, formerly instructor in physiology and pathology of the eye in the Ohio State University, died while on duty at the Base Hospital No. 14, Fort Oglethorpe, October 17th, from pneumonia following influenza.

PERSONAL.

Dr. G. A. Whitledge of Anderson, Indiana, has been elected a member of the American College of Surgeons.

Dr. E. DeWitt Connell, of Portland, Ore., is spending a portion of the winter in the East and South.

At the last meeting of the Ophthalmic Section of the New York Academy of Medicine, Dr. Arnold Knapp was chosen chairman

and Dr. Ellice M. Alger, secretary, for the year 1919.

Dr. Adolph O. Pfingst, of Louisville, Ky., has determined to discontinue treatment of ear, nose and throat diseases on January 1, 1919, and confine his practice to ophthalmology.

Dr. McCluney Radcliffe, of Philadelphia, recently read a paper on "Diseases of the Eyes in General Practice" before the Dauphin County Medical Society, Harrisburg, Pa.

MILITARY NOTES.

A son of Dr. Alexander Duane of New York City was recently killed in action in France.

Lt. Col. Casey A. Wood, for over a year on the staff of the Surgeon General, Washington, D. C., has received an honorable discharge from the Army. He will spend the winter in California engaged in literary work, especially in editing the final volumes of the *American Encyclopedia of Ophthalmology.*

Capt. W. E. Joiner, M. C., U. S. A., has been ordered to Base Hospital, Camp Fremont, Palo Alto, California.

Capt. Frank R. Spencer, M. C., U. S. A., is assigned to Hospital Unit 162, at present at Camp Lewis, Washington.

Dr. Thomas H. Curtin of New York sailed for army service abroad just before the signing of the armistice.

First Lt. John W. McCollon, M. C., U. S. A., assigned to Hospital Unit 162, at present at Camp Lewis, Washington.

Capt. Harry Vanderbilt Würdemann, M. C., U. S. A., appointed chief surgeon, eye department Base Hospital, Camp Lewis, Washington.

Lieut. Harry Boyd-Snee of South Bend has been promoted to the rank of Captain, and is located with the Base Hospital, Little Rock, Arkansas.

Dr. J. R. Sickler of Frankfort, Indiana, has been commissioned a captain in the Medical Corps and ordered to report at Camp Sevier, South Carolina.

Of the seven ophthalmologists of Jacksonville, Florida, three are still in the service. Major Norman N. Hegie was home for Christmas on a leave of absence.

Capt. W. R. Fringer of Rockford, Illinois, who has been stationed at Camp Joseph E. Johnston, Jacksonville, Florida, since August, received an honorable discharge December 10th and has resumed his home practice.

Lieut. Cowan of Philadelphia, formerly associated with the service of Dr. McCluney Radcliffe at Wills Eye Hospital, has been recently advanced to a captaincy, and is assisting Maj. Wiener at the School of Ophthalmology at Camp Oglethorpe.

Major Wm. T. Shoemaker, of Philadelphia, who went abroad as ophthalmic surgeon with the Pennsylvania Hospital unit, has recently been honored with the appointment of Consulting Ophthalmic Surgeon to all of the American Base Hospitals of England. It is expected that the promotion will advance Dr. Shoemaker to the grade of Lieut. Colonel.

The war fund for blinded soldiers and sailors founded by Mr. and Mrs. George A. Kassler of New York will supply the money needed to open an institute at Lille, for the blinded soldiers of northern France who wish to return near their homes in the territory recently evacuated by the enemy. This decision was taken on request of the French government.

Colonel Walter R. Parker has recently visited Philadelphia as the guest of the Ophthalmic Section of the College of Physicians, and gave its members a most interesting resumé of the work accomplished by the Division of Head Surgery of the U. S. Army. Col. Parker, as is well known, has been in charge of this division, and was permitted to visit Philadelphia by reason of a special dispensation of the Adjutant General of the Army. Following the presentation of Colonel Parker's paper, an informal supper was tendered him at the Union League by the acting president of the Section, Dr. S. Lewis Ziegler. Lieut. Col. George E. de Schweinitz was also present at the meeting of the Ophthalmic Section, and supplemented Col. Parker's paper by an interesting discussion of the important position the science of ophthalmology had been permitted to assume in the present war, as the outcome of the united efforts of a large staff of ophthalmologists now patriotically serving the country here and abroad.

MISCELLANEOUS.

Our San Francisco reporter says the flu is still raging but is beginning to ease up a little. He has seen some cases of acute glaucoma immediately following the influenza.

The Annales d'Oculistique announces that from January 1st, 1919, its subscription price will be raised from 25 to 30 francs per annum.

We are reminded by Dr. H. W. Hill of St. Paul that the masking of nose and mouth is not sufficient protection against the spraying of the influenza germ. He says germs that are coughed or fall into the eyes are carried directly into the nose by the lacrimal passages. Therefore it is highly necessary to protect the eyes against invasion.

Dr. W. S. Dodd, A. R. C., working at the Mejdel hospital unit, writes from Jerusalem at the American Red Cross: "The clinics numbered sixty to a hundred a day. Of course we had all classes of cases in medicine and general surgery, but by far the larger proportion of our patients were eye-cases.

"Of the 252 operations that I did in less than seven weeks, 222 were for the eyes. This is the number of persons operated on, most of them having more than one operation, perhaps on all four lids. so that I really operated on 408 eyes.

"There were some cataracts, not more than would be seen in the same number of cases elsewhere, but trachoma and its consequences account for almost all of the eye troubles in this land. I set out to treat these cases radically and secured fine results when I could keep the patients long enough for a reasonable after-treatment. But even so, the number of eyes that can be saved from partial and total blindness is large and the economic value of each eye thus saved is enough to make the prosecution of this line of work of the greatest importance for the redemption of the land."

OPHTHALMIC LITERATURE

These lists contain the titles of all papers bearing on Ophthalmology received within the preceeding month. These titles are all in English, some of them modified to indicate more clearly their subjects. These subjects are grouped under appropriate heads, the succession of groups being the same from month to month. In the group the papers are arranged alphabetically usually by the name of the author in heavy-face type. After the subject of the paper (Ill.) indicates the number of illustrations. (Pl.) the number of plates, and (Col. pl.) colored plates illustrating the article. (Abst.) shows that it is an abstract of the original article. (Bibl.) tells that the paper is accompanied by an important bibliography. (Dis.) means that a discussion of the subject is published with it. Under Repeated Titles are indicated additional publication of papers already noticed. To secure the earliest possible notice writers may send copies of their papers, or reprints, to 318 Majestic Bldg., Denver, Colorado.

DIAGNOSIS.

Alger, E. M. Ocular Malingering. New York State Jour. Med., v. 18, pp. 487-492.

Dufour, M., and Gaudibert, J. Ophthalmology and the Use of the Ophthalmoscope in Northern Africa. (2 ill.) Ann. d'Ocul., v. 155, pp. 473-477.

Franke. Roentgen Ray Diagnosis. Heidelberg Ophthalmological Congress, August 5, 1918. Abst. Klin. M. f. Augenh., v. 61, 1918, p. 352.

Grimm, K. Diagnosis and Logical Treatment of Eye Disturbances. Klin. M. f. Augenh., v. 61, p. 144.

Koeppe, L. The Nernst Lamp and the Corneal Microscope. Graefe's Arch. f. Ophth., v. 97, pp. 198-270.

Examination of Eyes by Polarized Light of Nernst Lamp. Heidelberg Ophth. Congress, August 5, 1918. Abst. Klin. M. f. Augenh., v. 61, pp. 349-350.

Romunde. Ophthalmoscopy with Red-free Light. Nederl. Ophth. Soc., June, 1918. Abst. Klin. M. f. Augenh., v. 61, p. 139.

Snellen, H. Visual Acuity as Measure of Function of Retina. Nederl. Ophth. Soc., June, 1918. Abst. Klin. M. f. Augenh., v. 61, p. 141.

Vogt. Fundus in Red-free Light; Living Lens by the Slit Lamp (Spalt Lamp.) Heidelberg Ophth. Cong., Aug. 5, 1918. Abst. Klin. M. f. Augenh., v. 61, pp. 350-351.

Wätzold. Difficult Questions for the Military Examiner. Heidelberg Ophth. Cong., Aug. 5, 1918.

THERAPEUTICS.

Axenfeld. Intraocular Ray Therapy. Heidelberg Ophth. Cong., Aug. 5, 1918. Abst. Klin. M. f. Augenh., v. 61, pp. 353-354.

Berneaud, G. Milk Injections in Ocular Disease. Klin. M. f. Augenh., v. 61, pp. 303-323.

Nakamura, B. Study of Improved Collyria and Eye Washes. Nippon Gank. Zasshi, Jan., 1918.

Nakamura and Mukai. Iodin Therapy in Ocular Diseases. Nippon Gank. Zasshi, April, May, June, 1918.

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Harman, N. B. New Eye Spud. (1 ill.) Lancet, Nov. 9, 1918, p. 632.

Taylor, S. J. Local Anesthesia in Ophthalmic Surgery. Brit. Jour. Ophth., v. 2, pp. 607-611.

REFRACTION.

Greeff. Indication of Cylindrical Axis. Heidelberg Ophth. Cong., Aug. 5, 1918. Klin. M. F. Augenh., v. 61, p. 352.

Henker. Indication of Cylindrical Axis. Heidelberg Ophth., Aug. 5, 1918. Abst. Klin. M. f. Augenh., v. 61, p. 352.

Oblique Rays in Astigmatism. Zeit. f. Ophth. Optik., v. 6, pp. 75-88. Abst. Klin. M. f. Augenh., v. 61, p. 366.

Landolt, E. Refraction of Eye. Arch. de Oft. Hisp.-Amer., v. 18, pp. 487-490.

Landwehr, J. H. Recognition of Anisometropia. (2 ill.) Klin. M. f. Augenh., v. 61, pp. 231-252.

Lindner. Skiascopy of Astigmatism. Heidelberg Ophth. Cong., Aug. 5th, 1918.

Mitchell, L. J. C. Eyes and Ears of Soldiers in Egypt. Med. Jour., Australia, Nov. 2, 1918, p. 365.

Olsho, S. L. Properly Fitted Spectacles and Eye-glasses. Pa. Med. Jour., v. 22, pp. 140-145.

Pyle, W. L. New Instruments and Apparatus. Amer. Jour. Ophth., v. 1, p. 852.

Rosenhauch. Changes in Refraction After Eye Wounds. Heidelberg Ophth. Cong., Aug. 5, 1918. Abst. Klin. M. f. Augenh., v. 61, p. 337.

Roy, J. N. Vision of Negroes in Africa. Ann. d'Ocul., v. 155, pp. 513-527.

Salzmann, M. Dioptrics and Anomalies of Refraction and Accommodation. (Bibl.) Zeit. f. Augenh., v. 39, pp. 322-336.

Watts, H. A. Necessity of a Cycloplegic in Refracting Myopes. Jour. Amer. Inst. Homeop., v. 11, p. 655-656.

Wätzold. War Experience with Myopia. Heidelberg Ophth. Cong., Aug. 5, 1918. Abst. Klin. M. f. Augenh., v. 61, p. 336.

Weiss, E. Discs for Mounting Trial Lenses. Zeit. f. Ophth. Optik., v. 6, pp. 49-52. Abst. Klin. M. f. Augenh., v. 61, p. 368.

Williams, E. R. Conservative Treatment of Asthenopia in Children. (Dis.) Amer. Jour. Ophth., v. 1, p. 860.

Wolff, H. Optics. Zeit. f. Augenh., v. 39, pp. 336-351.

Zade. Aerial Spectacles. Heidelberg Ophth. Cong., Aug. 5, 1918.

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Argañaraz, R. Nystagmography and Morphology of Nystagmus. Arch. de Oft. Hisp.-Amer., v. 18, pp. 469-486.

Bielschowsky. Bilateral Paralysis of Rotation; Treatment. Heidelberg Ophth.

Cong., Aug. 5, 1918. Abst. Klin. M. f. Augenh., v. 61, p. 348.

Dernehl, P. H. Strabismus Convergens. Early Recognition and Treatment. Wisconsin Med. Jour., v. 17, pp. 271-279.

Duane, A. Diagnosis of Motor Anomalies of Eyes. (Dis.) Amer. Jour. Ophth., v. 1, pp. 861-862.

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Marx, E. Fixation Under Different Conditions. Zeit. f. Psychol. u. Physiol. d. Sinnesorg., v. 47, pp. 79-96. Abst. Klin. M. f. Augenh., v. 61, p. 362.

Marx, E., and Trendelenburg, W. Precision of Position in Fixation of Eye. Zeit. f. Psychol. u. Physiol. d. Sinnesorg., v. 45, pp. 87-102. Abst. Klin. M. f. Augenh., v. 61, p. 363.

Repeated Titles. **Descomps, Enzière and Merle.** (v. 1, p. 620.) Jour. A. M. A., v. 71, p. 2027.

CONJUNCTIVA.

Bartels. Eye Diseases in Constantinople. (Dis.) Heidelberg Ophth. Cong., Aug. 5, 1918. Abst. Klin. M. f. Augenh., v. 61, p. 347-348.

Claussen. Trachoma as an Army Disease. Heidelberg Ophth. Cong., Aug. 5, 1918. Abst. Klin. M. f. Augenh., v. 61, p. 347.

Herrenschwand v. Parinaud's Conjunctivitis. Heidelberg Ophth. Cong., Aug. 5, 1918. Abst. Klin. M. f. Augenh., v. 61, p. 355.

Israel, J. P. Emphysema of Conjunctiva and Sclera. U. S. Naval Med. Bull., July, 1918, p. 473.

Kono and Ohashi. Bacteria in Normal Conjunctiva of Japanese. Nippon Gank. Zasshi, April, 1918.

Nakamura, T. Cause of White Color of Trachoma Granules with Use of Various Salts. Nippon Gank. Zasshi, March, April, 1918.

Terson, A. Examination of Upper Conjunctiva. Paris Méd., v. 8, p. 186.

Watanabe. Plasma Cells, Russell's Bodies in Ocular Disease. Nippon Gank. Zasshi, Jan.-Feb., 1918.

CORNEA AND SCLERA.

Dutheil. Pseudomembranous Pneumococcic Kerato-conjunctivitis with Purulent Keratitis. Ann. d'Ocul., v. 155, p. 528.

Eppenstein, A. Traumatic Etiology of Herpes Cornea. Klin. M. f. Augenh., v. 61, pp. 323-324.

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